UNITED STATES DISTRICT COURT

FOR THE WESTERN DISTRICT OF WISCONSIN

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GERALD BUSHMAKER,

Plaintiff,

-vs- Case No. 09-CV-726-SLC

RAPID-AMERICAN CORPORATION, Madison, Wisconsin
March 6, 2013
Defendant. 8:40 a.m.

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STENOGRAPHIC TRANSCRIPT OF SECOND DAY OF JURY TRIAL
HELD BEFORE MAGISTRATE STEPHEN L. CROCKER, and a jury,

APPEARANCES:

For the Plaintiff: Cascino Vaughan Law Firm
BY: ROBERT MCCOY
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JAMES HOEY
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For the Defendant: Rasmussen Willis Dickey & Moore
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Kansas City, Missouri 64114

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BY: MARK FELDMANN
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Appleton, Wisconsin 54912-0785

Also present: Donna Benson - paralegal

Lynette Swenson, RMR, CRR, CBC
Federal Court Reporter
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I-N-D-E-X

E-X-H-I-B-I-T-S

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(Call to order)

THE CLERK: Case Number 09-CV-726-SLC. *Bushmaker v. Rapid-American Corporation* is called for a conference and second day of jury trial. May we have the appearances, please.

MR. MCCOY: Yes. Robert McCoy on behalf of the plaintiff, along with Kevin Hanbury. And one of the attorneys is also here from our office again, Judge, James Hoey.

THE COURT: I remember him from the Pretrial

Conference. Good morning to all of you.

MR. HANBURY: Morning.

MR. MOORE: May it please the Court, Steve Moore and Mark Feldman for Defendant Rapid-American Corporation.

THE COURT: All right. Counsel, good morning to both of you as well. I hope you were able to use the snow day wisely. We obviously, or at least I hope it's obvious, we tried to leave hard copies of the order on Motion in Limine 23 on the desks. I issued it last night at 5:15. But at that point I was alone in the building, all the clerks had gone home, all the staff had gone home, so I couldn't file it last night and get it to you electronically. So we did it first thing this morning and then immediately discovered the typo.

It addresses 23. We put the text only on top of that. I think it speaks for itself. I don't have anything to add at this point. Certainly if we had had more time, I would have made it longer and more explanatory, but we've been down this road many times and frankly as far as I'm concerned, it's over.

Mr. McCoy, I don't know if you want to be heard further on that, but frankly I think I've heard and seen everything I need to. Having issued at least two written orders that address it, first the Final Pretrial

Conference order and then yesterday's order, I'm not seeing it, so I'm not going to let it in.

MR. MCCOY: Yeah, I thought the evidence, Judge, from the testimony that we had established, they had distributor lists that were readily available. I don't understand the lack of evidence. I mean it's for the jury to decide. They had plenty of ways to contact the people.

THE COURT: I'm not seeing it. No. You have given the Court nothing that would indicate the ability or efficacy of warnings. And again, it's not so much post-sale. It's hard for the Court to envision what's out there, and it's something I've commented on several time. It's not a criticism, but just an observation about why this is more difficult than usual for this Court.

You guys have lived this for years and you've got thousands of cases and you know a lot that this Court doesn't know and I think sometimes you assume too much and you don't put it into your filings or you don't make it know to the Court. And I don't think that's intentional, but I feel as if the Court is learning things in dribs and drabs, and as more becomes available to the Court, things become clearer, although never pelucid.

At this point -- and again, circling back and picking up with that observation, the Court felt compelled to go out and do some of its own research because from my perspective, I didn't have enough law in a timely fashion about 23. I think we all agreed that Wisconsin law is not entirely clear on this subject, but we gave the plaintiff a chance over the weekend, Sunday noon, to get us the proffer, and on a surface level the proffer looked good and so my initial ruling subject to input from the defendant was well, it looks okay.

And of course Monday morning, it's 8:30. We've got a jury waiting to be picked. I entertained brief argument and said listen, you know, we've got to pick the jury. You get me something in writing and I'll deal with it. I think the benefit of the snow day was that the Court then was able to be look at that. I'll be honest with you, I spent most of yesterday on a suppression motion or a suppression hearing in a criminal case. It's not like I spent the whole day on this. But I don't see how any of this testimony or any of this evidence would have led to a warning. I understand that there came a point where there are people talking about the dangers of asbestos and I understand that we've got the witness saying, you know, Philip Carey, here's things that you ought to do. Let's

talk to the engineers. Let's set up a program. Let's figure out what to do in the future.

Fine. That's good as far as it goes. But from the Court's perspective, I have seen no evidence that would tie that back to an actual warning. I have seen no indication there is a written warning that was proposed by anyone or how it could have actually been shared with purchasers or users of the product. And again, just saying that Asbestos Magazine and this other magazine is out there doesn't cut it. The dots have not been properly connected.

So Mr. McCoy, I understand your position, and if you're puzzled by the Court's ruling, I apologize. I think it's clear enough. I think it's more a matter that you disagree and think I'm making the wrong ruling. Well, you know, if it has to go up on appeal at the appropriate time, it will. But that's the Court's ruling. It's not coming in.

MR. MCCOY: Okay. That's on the warnings portion.

THE COURT: Correct.

MR. MCCOY: We mentioned on the unreasonably dangerous proof, that stuff --

THE COURT: Sure. And that actually raises a valid point. We had long testimony from the deposition

of Dr. Mueller or --

MR. MOORE: Mr. Mueller.

THE COURT: -- Mr. Mueller. Right. He never made it all the way through medical school 'cuz he ran out of money, if I'm recalling correctly.

MR. MOORE: Precisely.

THE COURT: About what was known in '85. Now to the extent that we need a limiting instruction to the jury that that is all still relevant to prove the dangerousness of asbestos, I agree, Mr. McCoy, that was the Court's pretrial ruling on that motion and it stands, and I don't think there's any true dispute that evidence to prove the dangerousness of the product is still relevant in proportion to what you need to show. I mean you can't overdo it. But if Mueller is the one, fine. If you've got someone else on that, fine. But yes, that's still in for that purpose.

And I guess Mr. Moore or Mr. Feldmann, I'll turn to you. Do you want a limiting instruction on the Mueller testimony in that regard at this point?

MR. MOORE: Yes, we do. Not at this point because I think we need to probably put our heads together and see what the nature of that would be and flesh that out a little bit, Your Honor.

THE COURT: Okay. Well normally, as you know,

courts give the limiting instructions at the time of the evidence. But I'm not -- I'm not telling you you have to.

MR. MOORE: I almost --

THE COURT: I mean if you want to make a catch-all limiting instruction at the end or simply put it into the written instructions at the end, we can approach it that way, too.

You guys want a minute to cogitate?

MR. FELDMANN: That would be good.

MR. MOORE: Yeah. I'm having trouble getting my arms around what's the effect of the Court's ruling, because I know Your Honor has a --

MR. FELDMANN: Let's talk about this.

MR. MOORE: Yeah, we'll talk about this.

MR. MCCOY: Judge, let me throw out just a couple comments on the ruling itself and its impact is what I'm talking about, the impact. Okay. There is some post-60 correspondence that talks about the pre-60 period that we would still intend to offer.

THE COURT: To prove the dangerousness of asbestos.

MR. MCCOY: To prove the dangerousness of asbestos. And also because it's pre-1960 conduct that's being talked about. It talks about what was being done

before 1960, even though the letter was written in '62. It says here is what we were talking about years ago.

THE COURT: Okay. And I don't have a problem with the topic under the Rules of Evidence. If it's Philip Carey's agents or people talking, then it's the statements of a party opponent. So I think that would get you over any hearsay hurdle. So far I'm not disagreeing with what you're telling me about the admissibility of that.

MR. MCCOY: But then I understand Your Honor's ruling to keep out the post-1960 conduct. That's what I understand and that would effect a lot of testimony that we've got designated --

THE COURT: True.

MR. MCCOY: -- which is a lot of what we spent time on yesterday. But what I'm saying is it makes it -- we've got certain depositions that I've got to think about and so on in terms of what we plan to present.

THE COURT: Sure.

MR. MCCOY: And it's thrown off a little bit, and I just don't know if we'll have until five o'clock today because of it.

THE COURT: No, that's actually a fair point and I want to respond to it in this fashion --

MR. MCCOY: I think we'll go to at least four.

THE COURT: If we have to stop early today, I will stop early today for at least two reasons: The important reason is I want everyone, particularly the plaintiff, to have a chance to respond to the Court's ruling and reorganize your evidence, and I don't want you guys to have to continue to do this on the fly. I think that this whole trial is sort of being done on the fly in some aspects, but that's neither here nor there at this point.

MR. MCCOY: That's asbestos litigation.

THE COURT: Well, it shouldn't be.

MR. MCCOY: Okay.

THE COURT: I think what we've got is a culture difference here between what this Court expects in its trials and what I'm getting from both sides here. And I'm not blaming the attorneys, I'm just saying we've never dealt with it before and it's hard for us to deal with it because we are so much more meticulous and OCD about the way we run our trials.

And so again, these weekend rulings and these morning rulings are in aphma to this Court, but we're trying to make it work. We're trying to be fair. And in that regard, to be fair, if we have to stop early today so that the plaintiff and the plaintiff's team can reassess and replan how it wishes to go forward, that's

fine. I'm not going to make you go on the fly.

The corollary to that is I don't think we lose anything in terms of the length of the trial because I've just kept out a lot of evidence, and that's not why I did it. If we had to go all of next week, we'd go all of next week. But, you know, if you need some time this afternoon either because you ran out of witnesses or because your team needs a chance to figure out how you want to go forward, we'll stop early. I have no problem with that.

MR. MCCOY: I mean I think we're good probably until about four o'clock. What I'm saying is that some of the stuff we had planned now needs to be edited and we're not going to be able to get, I'm sure, agreement of the parties today as to what's left or --

THE COURT: That's fair.

MR. MCCOY: -- be able to present to Your Honor a dispute before the end of today on the changes.

THE COURT: That's fair.

MR. MCCOY: And we've got -- we've got the Mueller dep -- a second Mueller deposition that has been prepared, I think with no post-60 stuff in it.

MR. MOORE: That can go in.

MR. MCCOY: Right. We've got worker's comp claims, which we can figure out which ones are '60 or

before. That's easy to do.

THE COURT: Is the doctor coming, the treating physican?

MR. MCCOY: Yes. Not today. We've got him for -- is it Friday, Kevin?

MR. HANBURY: Yes.

MR. MCCOY: I believe Friday.

THE COURT: Okay. What about the pipefitter?

MR. MCCOY: The pipefitter is here. That's what I'm saying.

THE COURT: Okay.

MR. MCCOY: He'll take awhile. And we also have the Humphrey deposition. So I'm just thinking it's about an hour.

THE COURT: Sure.

MR. MCCOY: With the exhibits, maybe half an hour.

THE COURT: But let me be clear, Mr. McCoy. You're sitting here sort of on the fly trying to figure out how much evidence you've got here today. Because of the Court's ruling, whatever you've got today is fine. I'm certainly not going to hold you responsible for running out of evidence today when you get a court ruling at 8:30 saying that a lot of what you thought was coming in is not. Okay? That's on the Court. That's

not on you.

So let's go as far as we can today. When you run out of evidence, let me know. We'll stop for the day and that's not a problem. Okay?

MR. MCCOY: Okay. Thank you, Judge.

THE COURT: Was there anything else before we bring the jury in?

MR. MOORE: I thought the temperature in here was a little high, but it seems like it's cooled down.

THE COURT: You too? I thought it was just me.

MR. MOORE: Okay.

MR. MCCOY: Judge, just for what's coming then, Dr. Brody, who has come in from North Carolina who managed to get here somehow yesterday, at least to Chicago and we got him here this morning, he will be the first witness. He's got a slide presentation.

THE COURT: Right. You mentioned he likes to use the old school slide screen. That's fine.

MR. MCCOY: He's given this presentation maybe 500 times.

DR. BRODY: Several times.

THE COURT: I'm sure it will be impeccable and polished.

MR. MCCOY: Mr. Moore has seen it, I'm sure. But the point of it is is that he will probably go up

and he's got his laser pointer.

THE COURT: That's fine. But let's be clear. To the extent that he's pointing with the laser, I'll leave it to you to make your oral record on the transcript.

MR. MCCOY: Yes.

THE COURT: If you need to articulate verbally what he's pointing at, feel free to do that. I'll let you be the master of the presentation. I'll let him. If he's done this a lot and he's been in court a lot, I'm sure he knows what to do and how to do it. So I'm not going to micromanage that.

Like I said, we try to stay out of your way. So once you guys start, we'll just let it go.

MR. MCCOY: Okay. That's basically exactly what I was going to say.

THE COURT: That's fine.

MR. MCCOY: That's how he does it.

THE COURT: Do you need the lights down lower for that? We can do that.

DR. BRODY: If it's possible.

THE COURT: It is. But let's circle back to getting the heat down. It's a constant problem. In Courtroom 250, Judge Crabb keeps it like a meat locker. It's about 40 degrees in there, and frankly, I think we

could use some 40 degree weather in here. If there were windows, we'd open them. But --

MR. MOORE: I thought it was just me, Your Honor, so...

THE COURT: Well, I'm higher than you in the room and I'm wearing a polyester robe, so take what you're experiencing and double it. That's what it's like for the Court.

MR. MOORE: Can I use the facilities before the jury comes in?

THE COURT: Sure. We'll start at nine.

(Pause 8:53-8:57 a.m.)

THE COURT: All right. Counsel, let's go back on the record. Mr. McCoy wanted to raise one more point before we brought in the jury.

MR. MCCOY: Yeah. One other item just came up. Mr. Ferriter is here. He's the pipefitting witness. I didn't know if you guys had objection to him staying in for Brody.

MR. MOORE: I can't imagine there would be any impact, Your Honor.

THE COURT: Okay. Well, I'm not going to sequester him unless you ask me to.

MR. MOORE: I can't imagine.

THE COURT: Sure. Let's let him watch.

MR. MOORE: Sure. Absolutely.

MR. MCCOY: And the other item I wanted to ask about, Judge, was the stipulation and/or instruction, however you want to call it, on the Rapid liability that's been tendered by the plaintiffs for the pre-1967 period?

THE COURT: I thought that the way I left it was you guys were going to meet-and-confer and if they agreed to it, fine. If not, then I needed some kind of response and objection.

MR. MCCOY: I think we confer every day.

THE COURT: All right. Well, then let me ask.

MR. MCCOY: We didn't get a response.

THE COURT: What is Rapid's response to the proposed stipulation?

MR. MOORE: Your Honor, I'm in the position where I believe I can agree to a stipulation. I want to check the exact terms of it. The jury -- I think we can even wait until or you can give an instruction to that effect at the end.

THE COURT: Sure. Well --

MR. MOORE: I'm not going to sandbag him on this issue.

THE COURT: No, he won't let you.

MR. MOORE: Obviously.

THE COURT: But I'll put the ball back in Mr. McCoy's court, but obviously I want you to deal with that so that Mr. McCoy doesn't have to fret about that.

MR. MOORE: I think it's -- I tend to agree with Mr. McCoy this is purely a legal issue; that the documents could be presented to the Court. You saw the chart that was presented in opening statement. You have a copy of it. So I think it comes down to a legal issue and I think we can enter into a stipulation to that effect.

THE COURT: Okay. Well, if not, I want to know before end of business Thursday so that we can make sure that the jury's got it before the plaintiff is prepared to rest.

MR. MCCOY: I was going to say, Judge, we'd like to have it given no later than Thursday.

MR. MOORE: Fair enough.

THE COURT: I agree. I think you're entitled to that. So if you guys can't agree on the wording, let me know not later than tomorrow morning and we'll do it the way that I do disputes over protective orders. It'll be like baseball arbitration. You each give me your proposal and I pick one without changing it.

MR. MOORE: Fair enough. Regarding the limiting instruction Your Honor asked about, I think we

would prefer to have it done at the time that the jury instructions are read.

THE COURT: Okay. And I think Mr. McCoy is entitled to be heard on that, too, but if you don't want it now, I'm fine with that. And if you'd like to propose a particular instruction, again sticking with this Court's OCD keep-it-in-front-of-you nature, the sooner you can get the language to me and Mr. McCoy, the sooner they can respond to it. If everyone agrees, I'll give it as offered. If they disagree, I'll referee that dispute. But the sooner you can get me some language, the better for everyone.

MR. MOORE: Thank you, sir.

THE COURT: Are we ready for the jury then? Mr. McCoy?

MR. MCCOY: Yes, we're all set for our jurors.

THE COURT: Okay. Let's bring in the jury. I assume we've got them all.

THE BAILIFF: We do.

THE COURT: All right. Always a good start to the day.

(Jury brought in courtroom at 9:00 a.m.)

THE COURT: Everyone please be seated. Ladies and Gentlemen, welcome back. I hope you used your snow day to good effect. I will confess that when I got up

yesterday morning and saw no snow, I thought that perhaps we had panicked for no good reason. But for better or worse, then we got all that snow last night or yesterday afternoon. So I think it was a right decision to make and it certainly has not put us behind in terms of the calendar I predicted to you when we did the jury pick.

So with that, we're ready to go. Mr. McCoy, who is your next witness, please?

MR. MCCOY: Actually it will be our first living witness will be Dr. Arnold Brody.

THE COURT: All right. Dr. Brody.

ARNOLD BRODY, PLAINTIFF'S WITNESS, SWORN,

DIRECT EXAMINATION

BY MR. MCCOY:

Q I'll let you make sure you've got some water ready up there, Dr. Brody.

A Okay.

Q Let's begin by introducing yourself to our jurors and give them your full name and spell your last name for everybody.

A All right. My name is Arnold R. Brody. B-r-o-d-y. I'm a Professor Emeritus in the Pathology Department at the Tulane Medical School.

Q And where do you presently work out of?

A So, I live in Raleigh, North Carolina. My wife is on the faculty there. I was there at the end of my career. And so I still carry out research with colleagues at Tulane University but live in Raleigh, North Carolina right now.
Q Can you briefly tell us about your educational background.
A Um-hmm. So, after high school in New Hampshire, I went out to Colorado to do a bachelor of science degree in zoology. That's the study of animals. I then went to the University of Illinois -- I know one of your favorite places around here -- went to the University of Illinois to get a master of science degree in anatomy. That is human anatomy; animal anatomy. That's where we learn how all of our parts fit together and function.
Then I went back to Colorado to do a Ph.D. That's a doctorate in cell biology. Every living thing is made of cells. We need to understand how cells function. Every disease has a target cell from which that disease develops. My focus is on lung cells.
Then after the doctorate, I did three years of study at another one of your favorite places, that's Ohio State University. So I was there for three years. And then I started my academic career.
Q All right. We've got here as Exhibit No. 36 a copy

of your curriculum vitae. Is that -- did I say that right?

A Yes, curriculum vitae. It's a resume. That's right. This one is a little out of date, but it's close.

Q Within a few months?

A Within a couple years.

Q Couple years. Okay.

A I'll send you a new one.

Q All right. You've done some work in asbestos specifically; right?

A For decades. Sure.

Q Okay. And is that part of what you've written on in the past?

A Yeah. Exactly. I mean I started my academic career as an Assistant Professor in the Pathology Department at the University of Vermont in the medical school, and while I was there, I met Dr. Wagner his name is. W-a-g-n-e-r. It looks like Wagner, but he's from South Africa and pronounces it Vagner.

He had established in 1960 that asbestos causes mesothelioma, this cancer that's caused only by asbestos. But he had developed what's called an *animal model* of the asbestos diseases: Asbestosis, scar tissue formation, lung cancer, and as I say, mesothelioma. He

saw the work that I was doing at the University of Vermont and he invited me to come and work with him.

So that was 1974 that I worked with him in Wales in the United Kingdom. That's when I started my interest in research in asbestos disease and have been publishing in that area ever since.

Q In terms of your training that you've had and experience, why don't you give us a brief summary of the important parts that concern asbestos diseases.

A Sure. So, obviously at the beginning, as I say, that was the University of Vermont. That's the real beginning where I started as a faculty member in the medical school; worked with Dr. Wagner. And then I was asked to take a position, I was offered a position at the National Institutes of Health. This is a government agency that's composed of about 25 different institutes. You've probably heard of the National Cancer Institute; National Heart, Lung and Blood Institute; National Institute of Allergy and Infectious Disease. The most recent one is the National Human Genome Institute. So those all make up this government agency, National Institutes of Health.

I was the head of the Lung Pathology Laboratory. Pathology is the study of disease. So I was the head of the Lung Pathology Laboratory at the National Institute

of Environmental Health Sciences. That's again one of these government institutes that make up the NIH. So I was the head of the Lung Pathology Laboratory. I was the head there for 15 years. That was from 1978 to 1993. So that's 15 years.

That's where I did a lot of the basic science research to understand where asbestos goes in the lung when it's inhaled, how it injuries various cells of the lung, how the asbestos causes the various diseases that it is known to cause. I've named three of them here already today.

In 1993, I was offered a position as a full professor in the Pathology Department at the medical school at Tulane University in New Orleans. Great opportunity. I took that position.

In 1999, I was promoted to Vice Chairman of the Pathology Department in the medical school. For those almost 14 years that I was there, I taught in the medical school. I taught medical students about lung disease and asbestos disease and diseases caused by inhaled particles. I worked with my medical colleagues there and published a long series of papers now getting into the areas of the genes that control these lung diseases.

The cancers are genetic diseases. Asbestos is a

carcinogen, a cancer-causing agent. All the asbestos varieties are. And we learned how asbestos causes the genetic injuries that lead to these diseases. I was there right through Hurricane Katrina in 2005. The hurricane pushed a lot of people out. It pushed us to North Carolina. I finished my career as a professor in the Department of Biomedical Sciences and Molecular Biomedical Sciences at North Carolina State University. I retired from there in 2011, the end of 2011. And then in early 2012, I was honored with the position of Professor Emeritus. Emeritus means from merit, out of merit, by Tulane. I was honored with that position.

They asked me to start a new laboratory again there. I declined to do that, but am working with some young investigators that I actually hired when I was there who now have their own research money, working on some of the things that I started when I was there on the genetics of and controlling lung cancer. So we're actually doing experiments now to understand how cigarette smoke and asbestos have a multiplying effect in causing lung cancer. That's some research that we're currently involved in right now.

And so as we're sitting here today, I'm continuing to write about this work and am invited to speak in various places around the world, including courtrooms.

Q Including Madison, Wisconsin.

A That's right.

Q Okay. You're not a medical doctor; right?

A I'm a Ph.D.; that's correct.

Q How is it that you'd be teaching medical students?

A Well, that's not at all unusual. I mean go to any medical school, any of the medical schools from coast to coast in this country. University of Wisconsin I know very well. I've given talks there. I was -- I gave a talk in Milwaukee just a few years ago at the medical school there. It's not at all unusual for Ph.D.s like myself to be professors in medical schools.

We teach the basic sciences to the students before they go into their clinical rotations. Things like biochemistry, molecular biology, anatomy, embryology, the things they need to understand before they start their clinical rotations.

Q You mentioned something about speaking internationally. Is that normally on the topic of asbestos disease that you speak?

A Sure. Yeah.

Q Can you give us some recent examples?

A Right. Okay. Well, the most recent one was in London. Last October I was asked to give a talk to a group of scientists there, pathologists and scientists,

and I explained some of the most recent findings that we've developed on how asbestos causes damage to DNA. DNA damages a precursor to the development of cancer.

I've given several talks already this year, one in San Francisco, one in Los Angeles, again on my work. But over the years I've talked -- I was a visiting professor at the Medical College of Beijing for several weeks; I did a sabbatical at the Institut Pasteur in Paris. I've given talks in Mexico and Germany and Sweden.

Many universities across this country. I just mentioned Wisconsin. But I've also given talks in Harvard School of Public Health, for example; schools in Texas and California. A number of different schools across the country many times.

Q You mentioned something about doing research in asbestos diseases. Can you just briefly describe for us how you go about conducting that research?

A Um-hmm. Sure. So the concept of basic science research is to develop new knowledge; in other words, what don't we know about a problem that we need to solve, that we need to understand before we can develop effective treatments or cures. There are no effective treatments for any of the asbestos-related diseases.

So the National Institutes of Health supports

scientists like myself through the awarding of grants to the universities and to the scientists. So over the decades, I've been competing with the great scientists in this state and across the country where we submit applications, explain the research that we want to do, what is the question that I want to ask that we don't currently understand. Like, for example, we know that it takes genetic damage to cause lung cancer and mesothelioma, but we don't know just which genes need to be damaged. I can give you a list of genes that I expect to be damaged, but we don't know in a given individual which set of genes is going to be sufficient for that person. And it's different for different people. That's one of the things that makes it very complex.

So I'll show you here today how asbestos damages DNA. But the new research is going to be able to explain how a given individual has a certain set of genes damage that causes the cancer in that person. That's the kind of new knowledge that needs to be developed. So when we submit these applications, we're competing with the great schools across the country, and about 10 to 15 percent of those applications get funded, so it's a very competitive environment.

My work was supported to answer these questions

without interruption through my entire career. I'm currently working with the investigators that I told you at Tulane who now have their own research dollars to carry out some of these very important questions. That's what the National Institutes of Health does. It supports the basic science, the research that's going on in this country, particularly in the biomedical sciences.

Q Dr. Brody, is it fair to characterize your research work as being done at the cellular and molecular level?

A Right. So I talked to you about cells. I told you about -- I'll show you what these cells look like in the lung. But it's the molecular level which is where science is today, and that means your genes. These are genetic diseases that we're talking about. Even asbestosis, which doesn't require genetic damage, but it requires the activation of certain genes, genes that cause cells to make scar tissue.

Well, I can show you, we have a whole -- I have a whole series of papers that describes how asbestos causes the activation of specific genes that lead to asbestos, asbestosis, and that's the kind of basic science research at the molecular level and cellular level as well.

But, you know, to start this off, I started

studying the lungs of people with disease and we also use animal models, which are essential. Name any human disease, and I can pretty much guarantee that we'll have an animal model to use to understand that human disease, because you can't do these kinds of experiments that we do on people.

Q Is it fair to characterize the work you've been generally describing, like DNA research and so on, and these -- as examples of these cellular and molecular changes?

A Yeah. Exactly. Sure.

Q And is it fair to describe these kinds of cellular and molecular changes as the asbestos disease process?

A Well, they're part of the process. Sure. Exactly. The process starts with the inhalation of asbestos, damage to the lung, activation and damage of certain sets of genes, and each of those issues is a whole field of research in and of itself.

Q Now Dr. Brody, just so everybody is clear, my law firm has brought you here today to talk about this asbestos disease process in a general way and I haven't asked you to testify to anything specific about Gerald Bushmaker's asbestosis or lung cancer or his own diseases or what caused it; right?

A That's right. And assume -- you've asked me to

assume there's been a diagnosis of lung cancer and I'm going to explain how asbestos and lung cancer develop in the lung and if that's, in fact, what he has, then you'll know how it happened. But right, you've not asked me to talk about him specifically.

Q We have other witnesses who will do that. So, and you're not here testifying about the field of epidemiology.

A I am not.

Q So, is it basically fair to say that your testimony today is about how asbestos gets into the lungs and causes asbestos diseases?

A Exactly. That's what I've been teaching medical students and juries for years.

Q So based on the research that you've described -- well, let me -- I'll go back to these other things in a moment. Just go ahead. Based on the research you've described so far, have you put together a slide presentation for our jurors today?

A Right. I have a slide set that I use, that I use for medical students. I use some of those to explain to juries. When I gave a talk in London in October, I used some of these slides I have here today. If I'm talking about the disease mesothelioma, I have a set of slides I use. If I'm talking about the most current research

from my laboratory, I have a set of slides for that. So of course.

Q Okay. And besides talking about asbestos disease, have you published in the field?

A Well, so the simple answer is yes. But you should know that you don't get to be a professor in medical school and go on and carry out a career unless you can publish your work in the open medical literature so that anybody who is interested and who is in this field can see what you're doing. I mean I want my colleagues to be able to see what I'm doing and read about what I'm doing, so I have to publish in the peer-reviewed press and book chapters and things like that.

Q Okay. So we'll talk more about some other publications, more of your background a little bit. But let's go ahead with the slide presentation. I think we're set for that, so we'll go ahead and get that running.

A All right. May I step down?

THE COURT: You may, and the microphone is there for your benefit. I think it's on, the hand mike. So Doctor, if you want to pick up that mike as well.

THE WITNESS: Okay.

Q I think we're set to go. We just need to use the clicker and go ahead.

THE COURT: Why don't we turn off a couple of lights here and see what works. See if we get the ones over the screen.
Q Doctor, I'll just introduce --
MR. FELDMANN: Actually that would be great.
THE COURT: All right. Let's leave it like this.
BY MR. MCCOY:
Q So, what we've got here is a slide presentation that you put together. These are your slides; right?
A Right. Most of these are pictures that I've taken with various kinds of microscopes or they are diagrams from textbooks and things like that.
Q Okay. So I'll let you go ahead and explain to the jury the significance of each of these slides. If I've got questions, I'll ask along the way.
A Okay. Great.
Q So the first slide.
A All right. So this is obviously a diagram. I know you know where your lungs are, but I just want to remind you that when you take a breath, the air comes down this tube that we call the *trachea* or *windpipe*. You can actually feel the top of that. That's the top of your trachea.
When you take a breath, the air comes down into

these series of tubes that are called *conducting airways* because the air then gets conducted down into the lungs. And then in among the tubes are what are called the *air spaces*, and we'll -- that's where we exchange oxygen, carbon dioxide. I'll show you that in more detail in a minute.

And then this line, this black line that runs around the outside of the lungs, that's represents the pleura. P-l-e-u-r-a. Very thin membrane, I mean Saran Wrap thin membrane; wraps around the outside of the lungs. Makes the lungs airtight like balloons actually.

Now I use this diagram as a map. I'm going to show you where the different asbestos diseases develop. So if you're an asbestos fiber, like the red spot on my pointer, and you come down the airway, the first disease that you come to is lung cancer because lung cancer develops in the walls of the airways.

I'm going to show you these things in more detail in just a second, but just as a map, an overall map, lung cancer develops in the walls of the airways. Then in amongst the airways where the gas exchange develops or the gas exchange takes place, that's where the disease asbestosis occurs. So asbestosis is scar tissue in the lung from inhaling asbestos. Now --

Q Doctor, just -- I don't mean to interrupt.

A Please.

Q If it's okay. But is this asbestosis a different disease than lung cancer?

A No question. It's different in several different ways.

Q And you'll be getting into more explanation of that; right?

A I will. But that's a very good question.

Q Okay. I'll let you go ahead then.

A Okay. That's fine. Lung cancer -- you know, you can interrupt me all the time. It's fine. So lung cancer is a disease caused by genetic damage and it's uncontrolled cell growth. We'll talk more about that.

Asbestosis is caused by the activation of certain genes and its production of scar tissue. So the lung gets stiff and a person is restricted from taking a deep breath. So if a person has a restrictive lung disease, for example, that means they're restricted from taking a deep breath, that's asbestosis. That can be caused by asbestos disease.

Now in the pleura, there are two diseases. One of them is a cancer called *mesothelioma*, and I understand we're not talking about that today. But there's another disease in the pleura. There's a very thin connective tissue layer underneath the outside lining of the lung,

and when asbestos gets to that very thin layer, it can cause injury and scarring, and if there's scarring just underneath the pleura, then it's called *pleural plaque*. Plaque is a circumscribed scar. If there are extended scarring of the pleura, it's called pleural fibrosis. Fibrosis meaning scarring.

So those are the asbestos diseases of the lungs. We can then take a closer look at the lung and see where these things are developing. I use diagrams a lot, but it's very helpful to see what these things actually look like.

So I use what's called an *electron microscope*.

Q That is a long time ago.

A Well, how can you tell? Yes. In fact, I had this microscope for decades. They're very expensive, hundreds of thousands of dollars, and they last a long time. In fact, this microscope was a victim of Hurricane Katrina.

I can take a piece of tissue as small as a period at the end of a sentence or as big as this device I have in my hand; I can put the tissue into this door right in front of me and enter it into a vacuum inside this column. And at the top of the column, there's an electronic gun that produces a field of electrons. The electrons come down through the vacuum and strike the

sample that I've put in, whatever I've put into the column.

The electrons raster scan over the surface of the sample, and wherever the electrons go on this sample, I can collect them with this electron generator over here and the electrons have a very short wavelength and can be magnified tens of thousands of times. So I can look at the tissue magnified much greater than you can with a light microscope.

I use light microscopes all the time, but the magnifications aren't big enough to see a lot of the fibers and a lot of the cells. So in front of me then appears an image of whatever it is I've put inside this door, and then just off of the screen is a camera so I can take a permanent image of whatever it is I'm looking at.

So for example, if I cut a piece of lung tissue out, and you'll see the conducting airways going up and you'll see the pleura running around the top of the lung, and I cut this out and I put it into the microscope and take a picture of it, it looks like this.

So here is that lobe of the lung. You can see the conducting airways running up into the lung and you can now see the pleura that I've cut across, and you can see how thin it is. So now we can use this as a map and get

a little better picture.

So lung cancer develops in the walls of the airways. Here's one of the conducting airways. Here's the gas exchange area of the lung; looks like a big sponge, sponge for air obviously, not water. And then you can see the pleura out here; very thin membrane. And this is absolutely normal. This is what the lung should look like.

Now I want to talk to you for a minute about what are called the *defense mechanisms* in our lung. As we walk around, we're exposed all the time to bacteria, pollen grains, particles, a few asbestos fibers. We're always being exposed to these things. And most of us don't get sick from these things because we have very effective defense mechanisms. And they start with the nose hairs and the moisture in our nose and the back of our throat constantly trapping things and we swallow them or sneeze them out. But a lot of things go right past that and get down into the airways.

And we have a very effective clearance protective mechanism covering the surface of our airways. I'm going to show you what that looks like now. I'm going to focus on the microscope right on the surface of the airway; could be anywhere along any of our airways, and let's focus this right down on the surface. And I'm

going to fill the screen with what's in that red spot. So we'll just be looking at what's in the red spot.

Let's take a picture. And now you can see this says *Human Bronchiole*. Bronchiole is a small airway, and the surfaces of our airways are lined by these little hair-like structures. They are really not hairs at all. They're extensions of the cell surface called *cilia*, c-i-l-i-a, and they're constantly being in a wave-like fashion, synchronous fashion, so that if something lands on the surface of our airways, it's swept up to our mouth and we swallow or spit it out. That's going on all the time in all of us. And when I say all of us, I'm not only talking about people, I'm talking about rats, mice, dogs, cats, guinea pigs, giraffes, whales. I mean every air-breathing animal has these same structures, same appearance, same size. If a rat or a mouse went running by me right now, they'd be doing exactly what you and I are doing: Inhaling and exhaling the room air using exactly these same structures that I'm talking about right here.

Q Dr. Brody, these are your slides that you actually prepared by taking pictures under the microscope?

A Exactly.

Q And what's the magnification on this one?

A Okay. So in the lower right-hand corner, you can

see this line, and this is called a *size marker*. We're always interested in how big these structures are. When we start looking at asbestos fibers, you want to know how big and small the fibers are. And this is a 10 micron bar. You see 10 microns. So it's easy to see 10 microns. But that's at a magnification of about 10,000 times.

So the question is how big is a micron, and I can easily explain that. So if you just take your thumb and your forefinger and you make a little space, you can just barely see with your naked eye. So you can look through that space that you've made, which is just about a millimeter. So with a naked eye, we can see just about a millimeter in size.

Now if you take that millimeter and divide it a thousand times, what you've done then is you've made a thousand microns. Okay. So you can barely see a thousand microns because a thousand microns equals a millimeter. You can barely see a thousand microns. You obviously can't see 10. You can't see a hundred. You can barely see a thousand. But with the electron microscope, of course it's very easy to see 10 microns. And if we want to know how long these cilia are in your mind's eye, scan this bar up right here, and you can see these microns are about 8 to 10 microns long.

Now these cells that are lining the airways, some of them have cilia and some do not. You can see that. Can you see that some of them look kind of naked? Those cells make mucus. And you don't think much about mucus unless you have a bad cold or you're a heavy smoker or if your airways have gotten irritated like mine have by whatever. Maybe I'm getting a cold or dried out in the airplane. Whatever it is. I'm making some mucus. You'll excuse me. I'm clearing my throat. This is mucus made by these lining cells which is constantly propelled up to your mouth so you can swallow it or spit it out.

Now these cells, the nonciliated cells of the conducting airways, are the target cell for lung cancer. Every disease has a target cell, the disease from which -- I'm sorry -- the cell from which the disease develops.

The cells lining the airways that are not differentiated, that means they've not fully differentiated into the final cell type they're going to be, that's the cell type that is the target cell for lung cancer. So it's not the ciliated cells because they've already differentiated. You can't make a ciliated cell into a cancer cell. But the cells surrounding it: The mucous-producing cells, the cells

that are going to be mucous cells, the cells that are going to be ciliated cells, those are the target cells for lung cancer.

And I told you lung cancer is developed in the airways. That's the target cell. I'm going to show you asbestos fibers. That's where the asbestos fibers go down into the airways. Okay.

Q Here's some -- something to help your mucus if you need it. I've got it close by for you.

A Thank you.

THE COURT: Let's take a quick break. I'll ask if we actually need the microphone. Why don't you put the microphone down and if we can still hear you fine, you won't have to carry it around anymore.

THE WITNESS: That's what I suggested earlier, but we'll see. How is that? Is that all right?

THE COURT: Can everyone still hear the Doctor okay? Let's keep going.

THE WITNESS: Now what we're going to do is we're going to go past the conducting airways. I showed you what that looks like and I explained that's what the target cells are. Now we're going to go out into the gas exchange area where asbestosis develops. So asbestosis develops out here in the gas exchange area, so that's where we're going to go next.

We're going to go past the escalator. I didn't use that term yet. So the mucociliary -- mucus and cilia, mucociliary escalator we call it because it escalates things up to our mouth and then you can swallow it or spit it out -- so we're going to go past the escalator and now we're at the end of the escalator where it empties out into the gas exchange area. So we take a breath, you and me and rats and mice, we take a breath and the air goes out into the air spaces and fills the air spaces.

About 20 percent oxygen in the room air. You notice when I cut this lung open, I opened up some little holes in the walls, and these little holes in the walls of the air space is where the blood runs. All the blood in our bodies has to run through our air spaces because it has to pick up the oxygen -- excuse me, has to pick up the oxygen that gets into the air spaces when we take a breath. So we take a breath and the oxygen in the room air diffuses into the blood that's running through the walls of the air spaces.

Now I think the easiest thing to do is think about this courtroom that we're sitting in as an air space. Take off the ceiling, look down on the floor and you see a carpet. This carpet looks like it's made up of a lot of carpet squares. If you think of each one of these

carpet squares as a cell, then you can envision what's actually going on in the floor of all of these air spaces. We have a series of cells, flat cells that make a carpet covering the surface of all of our air spaces. The difference here is that this carpet, if we're in an air space, the carpet goes up over the wall and into the next air space, so we have this complete carpet that covers all of our air spaces. And it's very thin. If I took a big saw and I cut through the room, I held up the cut surface, you'd see where the plumbing and the pipes and electricity and everything is running under the floorboards and in the walls.

Same thing in the lung. Cut it open and in the walls is running the blood and nerves and connective tissue. Same concept. We're going to talk about these cells that sit on the surface because these cells provide a pathway for the asbestos to get into the blood and the fluid flow of the lung, reach the pleura. And also it's underneath this carpet that the target cell for asbestosis lives. We'll talk about that in a second.

So we take a breath. Fill the air spaces. The oxygen diffuses out through the cells and into the blood that's running and the blood carries the oxygen to our brain and our fingertips and our toes. Meanwhile, the

blood cells that are giving up the oxygen and have carbon dioxide in them come back to the lung; release the carbon dioxide, you exhale. That's what respiration is all about. Exchanging oxygen/carbon dioxide. Same thing going on in all air-breathing animals.

Okay. Let's take a look at a single human air space and we'll take a close look at the carpet. Take the ceiling off, hang over a single air space, look down on the carpet, and it could be any one of these air spaces. So here is a single human air space. I'm outlining for you one of the carpet squares, but nature doesn't make squares very well. Nature makes smooth surfaces.

So this is a carpet oval, an oddly-shaped carpet cell. And then there's another oddly-shaped cell, another one, and then we have these sort of ovals here. These are all carpet cells. The big word is *epithelial cells*. Epithelia cells cover surfaces. So your skin is an epithelia. We call it epidermis.

So I'm standing on this epithelial surface here, and when you take a breath, we're going to -- I'm going to show you that some asbestos fibers land down here on the carpet. You might be wondering what these black holes are. When you take a breath, sometimes you get a little more air in one room than the next, and we need

to immediately even the air pressure in each air space, so nature has brilliantly drilled some holes in the wall. So if I took a big hole -- I'm sorry. If I took a big drill and I drilled a hole into the next courtroom, it would be like nature having produced these pores that allowed air to be instant equal pressure in all of our air spaces.

And it's fascinating. What's fascinating for me to learn and see this, you and I have maybe half a dozen of these holes in all of our air spaces, but horses and dogs that run very fast, they have multiple holes because they are constantly moving more oxygen and carbon dioxide than we could ever move in our lungs. So these are differences that are fascinating to learn among the different animal types.

Okay. Let me just say a little bit of word about these interesting looking cells here. They have little bumps all over them. The big flat cells are called *Type 1 epithelial cells* and these smaller cells with the bumps all over them are called *Type II epithelial cells*. If the Type 1 cells get damaged by asbestos or infection, these smaller cells start to divide and take their place. So we have a repair mechanism in every one of our air spaces.

Okay. One more line of defense and then we'll talk

about asbestos. I'm going to focus the microscope right down on the air space. Remember the cell with bumps all over it? I'm going to focus right down in the human air space. Here is the cell with the bumps. Now you can see I'm focused right down on it. Here is the carpet down here.

Now we have these two added cells. There's this one with kind of ruffles all over it, and then there's another -- same cell type, but this one is stretched out. It has a tail end and it has a couple of what are called false feet. If you ever had a biology class, you might remember pseudo pods or false feet. This cell I caught in the act when it was going after this pollen grain right here.

Now this lung once belonged to somebody who was killed in a motorcycle accident. I was on the medical examiner's autopsy call. I went in and prepared this person's lung within hours of death. We have chemical fixatives that stop all the life processes in these cells in very life-like conditions so we know just what they look like when the cells were living. When this person was rolling along on his motorcycle, he obviously was inhaling a lot of things, a lot of things got caught in his nose and in his trachea that got swept up by the cilia. But one thing we know got past the cilia is this

pollen grain right here, and it landed on the carpet. We don't want any kind of foreign particles sitting on our gas exchange surfaces.

So we have these cells called *macrophages*. Macro means big, phage means eater. Cells that are big eaters of the lung. They patrol our air space surfaces. They can detect the presence of foreign particles with chemical detectors called *receptors* on their surface. In my laboratory, we discovered the chemical signal that attracts macrophages to asbestos fibers because we certainly don't want -- we don't want pollen. We don't want anything -- we certainly don't want toxic asbestos fibers sitting on our air space surfaces, and in fact, that's part of the disease asbestosis I'm going to tell you because when these macrophages try to wrestle with asbestos fibers, it causes the death and activation of those macrophages and that adds to the development of the disease. I'm going to show you how that happens.

So what's going to happen is this macrophage is going to pick up the pollen grain and it's going to migrate onto the escalator, and every time you swallow, you swallow a few of your friends, these macrophages. Because each one of us, in rats and mice and guinea pigs and giraffes and horses, we all have about one or two macrophages in every normal alveolar space. If you

smoke, you have hundreds because the macrophages are constantly trying to clean up the stuff from cigarette smoke. Assuming we don't smoke, you have one or two macrophages in every air space, and they would be, if you think again about this courtroom, the size of this courtroom, it would be about the size of you or me standing in a courtroom, actually lying down on the carpet. That would be about the size. And when a macrophage or two then does what it's supposed to do, it gets up on the escalator; clears out. We're constantly making new macrophages in our bone marrow. We have macrophages in all of our organs: The liver, the brain, the GI tract. They get formed in the bone marrow. They travel through the body and they climb out of the vasculature, out of the blood flow wherever they're needed: Air spaces, liver, brain, wherever they're needed. And they're ready to go and fight infection or whatever might land in those organs.

Okay. So now you've seen all of the cells you need to see to understand what happens when asbestos gets into the lung. So we can talk about asbestos.

Now this is chrysotile asbestos. And chrysotile asbestos is about 95 percent of the world's use. Now there are two other asbestos varieties that make up just about the other 5 percent. You can see how chrysotile

is spelled. That's about 95 percent. The other is one crocidolite. C-ro-c-i-d-o-l-i-t-e. Crocidolite. And then the third one is amosite. A-m-o-s-i-t-e.

So crocidolite and amosite are a different mineral form of asbestosis, different than chrysotile. All of the asbestos varieties cause all of the asbestos diseases. There is no safe asbestos. They all cause all of the asbestos diseases that I've talked about. I typically -- I've used them all in all the work I've done. I've use chrysotile most because it's 95 percent of the world's use and there are more questions being asked about chrysotile than the others. So I use chrysotile.

But when I talk about asbestos, I'm talking about asbestos. That's a general principle because it does all of the things. All the asbestos varieties do all the things that they all do.

Now the nature of chrysotile is that it tends to fracture, and you can see some of these different shapes and sizes. Notice there's a size marker down here with a 1.0 micron. So this is a one micron bar. Remember you can barely see a thousand microns. But it's easy to see one micron at a magnification of 4,300 times. That's what that says. 4,300 times magnification. It's easy to see a micron. It's easy to see that some of

these fibers start at a micron thick and then fracture down to a half and then fracture again to even less. Some of them get down to tenths of microns. A large variety of shapes and sizes. That is the characteristic of asbestos. Not only chrysotile, but crocidolite and amosite as well.

Infinite variety of shapes and sizes. That's the concept. Chrysotile tends to break down more. It's more easily broken down. It's more easily transported out of the lung and more easily transported to the pleura. But understanding that all of the fibers are involved in diseases, I'm just telling you some differences about the way chrysotile is handled in the lung. We can talk more about that if you're interested.

Okay. Now I'm going to show you a couple of experiments from my laboratory. One of the very first questions that we ask, sounds kind of simple and mundane, but when I started my work in the 70s, we knew that asbestos caused all the diseases we talked about and we knew that obviously that fibers get into a lung, but we didn't know where they go in the lung. If you would have -- I went to look in the literature and asked well, do the fibers land on this epithelial carpet that is required for movement of oxygen and carbon dioxide? Nobody knew.

So I started a series of experiments where we used what are called *exposure chambers*. Six feet high. Four feet wide. An asbestos generator at the top of the chamber; makes it dusty in the chamber. With the animals, typically rats or mice, which we know develop the same diseases as people: Asbestosis, lung cancer, mesothelioma; same target cells in the animal models. Same disease. Same place in the lung.

So, now I can't just say that. I have to prove that to my peers. I don't get to publish the animal model unless I prove to my peers that I can answer the questions that I want to ask. I can't answer everything I'd like to ask about with the animal models. I can't tell you, using an animal model, how much asbestos it takes to cause the disease in people by using animals. You can't do that. Nobody should try to do that because you can't do that.

So that's an example of a question I'd like to know the answer to, but you can't learn that by using animals. But we certainly can learn where the asbestos fibers go in the lung; how they interact with the various cells of the lung that I've told you about. And let me just show you a little bit of that.

So we make an aerosol of the asbestos. The animals can be exposed for an hour; take them out of the

chambers; give the animals an overdose of the anesthetic -- overdose means they don't wake up from that -- and then any asbestos that we see in the lung must have landed there during that first hour. It's the only way you're going to get asbestos fibers into the lung of these exposed or these experimental animals.

So let's take a look. So here is one of these regions in the lung now. You remember -- you recognize the end of the airway where it opens out into the air spaces into the little rooms. Here is the blood flow in the walls, just like you saw in the human lung.

This is from an animal, a rat exposed for a single hour. Now this is one of millions of places like this around the lung. Multiple animals done, multiple papers published on this. I'm showing you one. One example.

So I'm going to focus the microscope right here and take a picture. And in this black hole right here is this black hole right here so you can see exactly where we are. We're going to look at the carpet right here. And what do we see on the carpet? A lot of the fibers now sitting on the carpet. And this is a 10 micron bar, so that means this fiber is about 10 microns long. And there's kind of a long curly one here and there's some straight fibers in various shapes and sizes. Remember the shapes that we saw here in the microscope, some of

them are long and some are short, some are curly and some are straight. Same thing sitting on the lung.

Here is a blood vessel running through here. If I cut it through, you'd see that little hole where the blood is running.

Now, here are the fibers sitting on the carpet. Now we know these fibers are toxic. We know they cause injury. And what happens when they do? That's what we were able to answer with the animal model. Remember, a person, you can see the disease in a person's lung. But that's typically decades after exposure. I'm going to explain how it takes so long. But those decades after exposure mean that something has been going on in those lungs for those decades. The only way you can learn that is by using the animal model.

So here are the fibers now sitting on the carpet. And what happens next? I wrote a paper called *A Month in the Life of an Inhaled Asbestos Fiber*. So, I mean it was kind of tongue in cheek, but really I was able to explain using these kinds of experiments. So the next group of animals you take hours later, then you take some days later, weeks later, months later and follow this process. Where did the fibers go? What did they do?

So here, already you can see that some fibers you

can see clearly and some you can't because they're covered by the carpet. You can see the fibers easily here, but you can't see them here because they're covered by the carpet. What we learn was that some of these carpet cells very actively come up over the top of the fibers and shove them under the carpet.

We can see that even a little better in this picture. This is another animal, one of thousands of places like this. Another experiment. Air space here. Another air space. Here is the continuous carpet. There's a little fiber bundle here. There's a little bit of a fiber going into the lung here. You can see the fibers here, but you can't see them here. Look at this fiber. It's completely covered by the carpet.

Now you may also have noticed these characters that look like doughnuts. This is what your red blood cells look like. Your red blood cells look like doughnuts because they have a depression, not a hole, but a depression in the center, and they are designed to have a very broad surface area so that they can hold a lot of carbon dioxide and oxygen.

Now this is the lung of a rat. But I said, this is what your red blood cells look like and mine because that's exactly the size and shape of yours, mine, guinea pigs, dogs, cats. I like to say giraffes because that

seems so odd, but they have the exact size and shape red blood cells as you and I. And these blood cells move through these small blood vessels called *capillaries*. And you can see that some of the vessel -- some of the asbestos is actually moving into the blood flow. And we published several papers showing that asbestos gets into the blood flow. And if it gets into the blood flow, it can go anywhere in the body.

So there are a couple things going on already. One is that asbestos is getting into this compartment underneath the carpet. And it's getting into the blood flow. I'll tell you the significance of that in a minute.

But the fact that it's getting into this compartment underneath the carpet; and we know that about 20 percent of all the fibers that get inhaled and that land in the carpet are taken up into this compartment.

Now what's in the compartment underneath the carpet? I told you you see that there's blood flowing there. You've already seen that. Remember, we're looking through the carpet here. Okay? The electron microscope is blasting those electrons right through the carpet here and you can see the blood flowing just like a pipe. Just think of a pipe underneath the floor here.

That's exactly what's going on. Okay?

Now what's going on in the carpet? So the asbestos fibers have landed on the carpet. The carpet cells respond to this foreign particle and come up over it and shove it under the carpet.

Now, you may have heard, or maybe you haven't, that we all have some asbestos fibers in our lungs. That's from the environment. It's what's called *ambient exposure*. We all have some fibers. Most of us don't have many, and many can be hundreds of thousands because that's not a lot. Because if you took my lungs and stretched them out, they'd cover the surface area of this courtroom. We have a huge surface area. If you sprinkle a few hundred thousand microscopic fibers on that, that's not a lot.

When I'm talking about a lot, I'm talking about trillion -- billions and trillions of fibers inhaled by a person who is exposed occupationally or environmentally to much higher concentrations that are in the background than you and I get. But where are our fibers? Sitting underneath the carpet in that space, and they'll sit there forever and not cause any disease because that's where we store things like that. Cigarette smokers just load that compartment with carbon and particles from cigarette smoking.

Now, in that compartment is the carpet cell for asbestos. It's a cell called the *fibroblast*. Fibroblast. And if you take your skin and you pinch your skin, hopefully it'll pop back. And it pops back because the fibroblasts that live in our skin are making connective tissue, elastic connective tissue. Elastic. That's exactly what it is. Elastic. If you take a breath, your lungs expand; you release the breath, the elastic tissue that the fibroblast make allow the lung to just collapse back to its normal condition.

Now when the fibroblasts are injured or when the cells surrounding the fibroblasts are injured, like if you fall down and scrape your skin, very likely you get a scar there if it's a bad scrape. I have some scrapes from barb wire when I was a cowboy. And those scars will last forever.

Scar tissue is stiff, is stiffer than this normal connective tissue. That's why when a person inhales asbestos and he gets enough asbestos into that compartment and he activates the fibroblast to make scar tissue, he can develop the disease asbestosis over a long period of time. It typically takes a long period of time for sufficient scarring to develop.

The scarring can start right away. We've shown in our animals that you can get scarring in a week just

from the hour exposure that we give them; high concentration, but enough to produce injury and scarring. So that's how we studied the scarring process. And we know that if you continue to expose the animals, the scars are not going to go away. They're not going to recede, they're going to keep getting worse. And in a person who's exposed to sufficient asbestos, enough fibers get into that compartment to cause the disease asbestosis.

Now the result of that is, I think I mentioned earlier, a stiff lung. The person is restricted from taking a deep breath. So if a person is exposed to asbestos, has the appropriate shadows on an x-ray, has shortness of breath, has what's called *restrictive pattern* where you can't take a deep breath, that's all typical of the disease asbestosis.

Okay. Now, the macrophage now are being called. The fibers are landing on this carpet. They're causing injury. The macrophages come in in bunches, and that's why I got into this issue of trying to understand the mechanism that attracts them. How do the macrophages -- they don't have eyes. How do they detect where the fibers are? Well, it turns out that there's a chemical signal, that I won't get into, it's a series of four or five papers that came from my laboratory that describe

the chemical signal that attracts the macrophages.

And here you can see a group of macrophages: 1, 2, 3, 4, 5. You can see that some of them are sharing asbestos with one another. Remember what the normal macrophages looked like? Ruffles and ridges and no holes. No holes in the membrane at all. Very compact. Here is what they look like when they're trying to tackle asbestos. Shrunken. Little holes in them. This one is not too bad a shape.

But these macrophages are dying. They've been activated and they're dying. I told you these fibroblasts that live under the carpet are making connective tissue. Well, they don't just make it spontaneously, they have to get the signals to make it. They have to be told to make it. And they're told by what are called *cytokines*. Cytokines are chemical signals that tell which cells communicate with one another. These macrophages make a series of cytokines that tells fibroblasts not only to divide and make more of them, but to make scar tissue. So at the same time the macrophages are coming in to clean out the asbestos, which they do, a lot of them do, they're causing the production of the proteins, these peptides, these cytokines that cause the fibroblasts to make scar tissue. So they're part of the disease process.

Now, this is what the lung looks like of a person with three asbestos diseases. Now this says severe asbestosis and pleural fibrosis. Now, this is so severe that when the lung of this person was cut across, you can see this white stuff? That's actually scar tissue. So you can actually see it with the naked eye, there's so much scarring in the lung.

Now the normal pleura should be very thin. Remember, I told you Saran Wrap thin. And this is fairly close to normal. But look at this. This is pleura. This is a pleural plaque. You see this area? Because a lot of the asbestos fibers that get into that space get carried to the pleura and they injure the fibroblasts that live just underneath the pleural membrane. And when those fibroblasts get activated, they make scar tissue. Same issue. And you get this thickening of pleural fibrosis or pleural plaque.

Now there's also a cancer. This is a lung cancer that's grown dramatically. It's made a big tumor. And that's not at all unexpected. When you have a high level of asbestos exposure, you have this severe disease; may or may not be a smoker, and you get a lung cancer. We'll talk more about that in a second.

Q Doctor, I have a question now.

A Uh-huh.

Q Are you saying this slide represents a lung that has three separate asbestos diseases?
A Exactly.
Q Can you just -- I wasn't clear. Where would the asbestosis be versus the pleural fibrosis?
A Okay. So you see this white -- so let's start this way. If I took a piece of this brown area here, this stuff here and I put it under a microscope, you'd see all the little air spaces and you'd see the red blood and you'd see the capillaries running through. So it would look like that sponge that you saw.

But if I took this part of the lung out, all of those spaces would be filled in with scar tissue and the walls would be thick. So you can't -- you wouldn't be able to have oxygen and carbon dioxide exchange. So these areas of big white -- and you can also see some black pigment in there. This guy was probably a smoker. Maybe he was a miner. And he's storing a lot of carbon and silica and things like that. So this is a relatively normal lung, this is a scarred lung, and then on the surface of the lung there's a plaque because it's circumscribed and then there's a linear pattern of scarring that called *pleural fibrosis*.
Q This is like a slice on a slide. Is that what we're looking at?

A Actually this is called a *whole lung*, and then also, as I mentioned earlier, there was a lung cancer. Now this started, the lung cancer started as a microscopic nodule that grew out dramatically and very quickly, and I'll show you how that happens because we're going to start talking about cancer here in a second and then I'll be finished.

Q But the lung cancer, is that just the thin line that's running along there?

A No. Actually there's a tumor that goes around like this and then you cut just a piece of it off so you can see that. But to answer your question, this is called a *whole lung section*. So what we do, and actually we did this in the laboratory, Dr. Wagner's laboratory in Wales, you take a person's lung from autopsy and you can take a very sharp knife and do a very thin slice and then you mount it on transparent paper so you can actually look through it and get a picture like this. It's called a whole lung slice.

Q Thank you.

A Okay. So let's see where we are. All right. So I'm going to review the diseases in diagram form and then I'm going to finish by talking about cancer.

How did the asbestos that landed on the target cells of the airway cause a lung cancer? That's what

I'm going to talk about. But first, here is a slide. These are called *Netter diagrams*. N-e-t-t-e-r. Dr. Netter has given us atlases of the human body in health and disease and we use Netter diagrams all the time lecturing in medical school. And here, Dr. Netter is showing us asbestosis. Scar tissue in the lung. He's showing us an x-ray where there are linear shadows. And then he also has cut the ribs away and he's showing us that on the surface of the lung are pleural plaques. So here are two of the diseases, asbestosis and pleural plaque.

And then Dr. Netter is now giving us -- showing us a lung cancer. It's fairly well developed. You can see that it started in the central regions of the lung and has now grown out into the lung, which is fairly typical, with a normal -- this particular individual has a normal pleura, thin shiny pleura. Okay. I have about five or six more slides and I'm going to talk about how asbestos causes cancer.

This is the cover of proceedings of a meeting I was at a few years ago and the topic of the meeting was how fibers cause cancer. Carcino, cancer; genesis, beginning of formation. So how fibers cause cancer. I think I said I gave a talk there. A lot of people were presenting information at this meeting. And I've showed

you cells, I've showed you what cells are, and some cells of the lung pick up these fibers. But you can't talk about carcinogenesis unless you talk about the molecular aspects. And I already told you that means your genes.

Cancer is a genetic disease. And I'm going to give you the simplest definition of cancer and then I'm going to explain what that means.

So cancer is the loss of control of cell growth. I'll say it again and I'll explain what that means. Cancer is the loss of control of cell growth. If I took a piece of your skin, I put it under a microscope, I can predict that about 10 percent of your skin cells are growing. You need to -- you're always losing skin. You need to make new skin cells to replace it. So about 10 percent. That's normal growth rate.

Your lung and your liver about 1 percent; very low background rate of growth of your lung and your liver. But every cell in the body, except red blood cells, need to divide and multiply at some time during their life cycle.

The airway lining cells have a low rate of about 1 percent. Very low background rates. And we've studied these various rates. We know that when asbestos lands on these air spaces, those Type II epithelial cells go

into a rapid up-regulation of growth. But when you stop exposing them, they go back to the normal because no cancer has formed yet. To produce a cancer, the growth rate continues until the cancer brings that person to the clinic.

Okay. Now, let's start again and keep it simple because cancer is a very complex process. Loss of control of cell growth. Humans have about 20,000 or so genes that make us what we are. If you look around, you see various hair color, eye color, skin color. That's a few things in what our genes do. Most of what our genes do you don't get to see; making this waterproof skin, making digestive enzymes. Things that we are doing all the time. That's what those 20,000 genes are doing.

About 100 of those 20,000 genes are called *growth control genes*. I told you the cancer is caused by loss of control of cell growth. Cancer develops when there are errors or mistakes, sometimes called *mutations*, in a set of genes that control cell growth. Okay? So a carcinogen causes damage to the DNA, DNA meaning your genes, the DNA of the genes that control cell growth. That's what carcinogens do. They damage genes that control cell growth.

Now one of the ways that we study that is by exposing animals. They get lung tumors. They get

mesothelioma. And you can go into those tumors and you can see what errors have been produced in the tumors. But if you really want to watch what's going on, we do other kinds of experiments called *in* vitro where we actually take the cells out of animals, people, and put the cells in a dish. You give them the right nutrients, the cells will grow. You can even form tumors in the dish. You can add the carcinogens. They damage the DNA and cause cancer in the dish. So by having these cells out, you can actually add the agent, suspected agent or known agent, and see what happens.

Here, for example, on the cover of this proceedings, are two cells. You can see one cell here. There's another one over here. Two of millions of cells in a dish. I'm showing you two of them. Fibers have been added. You can see there's a long fiber here and some short fibers. You can add fibers. You can add cigarette smoke. You can add components of cigarette smoke. We've done all these things, and see how they interact with the DNA.

Now, you see the center circle in the cell. You notice that the fibers have been excluded from the center circle. The center circle is called the *nucleus* of the cell and that contains all of your DNA. Pick out a cell: Lung cell, skin cell, all of the cells, airway

cells, they have that center circle called the nucleus and all of the DNA is in that. Our of our DNA is in that nucleus.

Now, sometimes it's confusing to think about well, if all of our genes are in that one nucleus, why is the skin cell different than the lung cell? And the answer is simple. The answer is our genes are like a symphony orchestra. The cells in our skin, only the DNA, only the genes that are making skin cells are playing. The genes that make lung proteins and digestive enzymes are quiet. You go to the stomach, take a cell out, look at which genes are playing, they're the ones that are making digestive enzymes and the skin cells are quiet. Okay? And that's the same story throughout your body.

All right. So here, notice how the fibers, the carcinogens are excluded from the center circle from the nucleus. And that's good. You don't want the carcinogen interacting with the DNA. So we have this protected membrane. But scientists have known for a long time that when cells divide, they lose that protective membrane.

So in my laboratory, we added fibers to cells that are dividing. Let's see what happens. Here are three cells: One. Two. Three. The two cells on the outside are not dividing. The DNA has been staying blue in the

nucleus, the center circle, so you can see the DNA. DNA just means your genes. Your genes make up all of your DNA.

The cell in the center has received a signal to divide. It could be that it was a skin cell that got scraped away in a fall and the surrounding cells needed to repopulate that space and so they got a signal to divide from the blood. Or it could be that I added a growth hormone to the dish. Whatever the reason, the idea is to make another cell, two cells just like the original. And the way we do that is by condensing our DNA into these white threads called *chromosomes*.

Chromosomes are bands of condensed DNA. Let's see what our chromosomes look like. See, what we're going to do then, once we get them into the chromosome, we're going to make perfect copies of all of our chromosomes. And if you look at our chromosomes, this is human karyotype which means the chromosomes spread. Humans have 23 pairs of chromosomes. You got one of the pairs from your mother and one from your father. They're all numbered. You can see these light and dark bands on each of the chromosomes. That represents specific areas of genes.

The important point to remember about this slide and this concept is that every one of our genes must be

on the correct chromosome and in the right place on that chromosome. There's no mixing and matching allowed when it comes to where our genes are located. If the gene is in the wrong place on the wrong chromosome, it will not function correctly.

A lot of examples. I mean we're all familiar -- we're all familiar with Down Syndrome. Down Syndrome occurs when there are mistakes or misplacements of genes or parts of Chromosome 21. It's very clear. That's a well-known example.

Now let's see what happens when things go as they should. Normal cell division. Signal to divide. The chromosomes are formed. Now they replicate. And if you have what's called *faithful replication* where each gene is in the correct place on the correct chromosome, you get then what are called *daughter cells*. And here you have the two daughter cells, just like the first one, and life goes on. And you're constantly doing this all the time in your body.

Now let's see what happens when you put asbestos into the story. Now let's think about these airway lining cells. And the airway lining cells you saw extend out, all the way out into the bronchials, all the way across the lung. And asbestos fibers are constantly landing on those bronchials and bronchi. And most of

the fibers get cleared up out of the lung, but some of the fibers are picked up by the airway lining cells and then have access to the DNA of those cells.

Now, what happens if they do? Well, here you can see a normal cell. No fibers. Half the chromosomes go to one side, half to the other side, and we'll have two daughter cells just like the original.

Now crocidolite asbestos has been added. You can see a long fiber. So this cell from this side to this side is about 40 microns across. You of course can't see 40 microns with your naked eye. These are taken with a microscope. And you can see this fiber is about 30 microns, 20 microns, some small fibers, and these have aeromeds on them because some of the fibers have DNA bound to the surface of the fibers.

Okay. I just wanted to check. I have one more slide after this. Okay. So what's happening is that there is DNA bound to the surface of the fibers. That means that all of the DNA is not where it's supposed to be, resulting in this condition called *aneuploid*. Aneuploid means abnormal chromosome separation.

Now these aneuploid cells -- and again, aneuploid is produced by all the asbestos varieties and by cigarette smoke. Aneuploid cells are not cancer cells. But the door has been opened. Let me tell you how the

door has been opened and then I'll finish. I'll have a summary slide.

I told you that humans have about 100 or so growth control genes. I also told you that every gene has to be in the right place in the right chromosome to do what it's supposed to do. Of the 100 or so growth controls, let me tell you about two of them because I've worked on them in my laboratory and I know something about them. One of them is called p53. p53 was a Molecule of the Year in 1993. Molecule of the Year means it gets its own cover on Science Magazine and it really is very significant in understanding human disease.

This is called a *tumor suppressor protein*. That's what the "p" is. And 53, don't worry about that. So p53 is a tumor suppressor protein. When a cell again has DNA damage, like in these aneuploid cells, as you can see here, p53 gets activated and stops the cell from dividing. For a cell to become a tumor cell, it has to pass the mistakes on to the daughter cells. If the cell is not dividing, it can't pass the mistakes on. That's why it's called a tumor suppressor protein. Stops the cell from dividing.

Now what if, in this DNA that's sitting right here, is a p53 gene or we have several like it, but what if the p53 was sitting there? Well, it's certainly not

where it's supposed to be in the normal array of genes on our chromosomes. It's not going to work. So the cell then is more likely to go -- to continue on through the growth cycle.

Tell you about one other thing. They're actually a series of genes, they're called *death pathway genes*. There's actually a big word for that that you might have heard of, it's called *apoptosis*. A-p-o-p-t-o-s-i-s. Apoptosis meaning programmed cell death. You go out in the sun and you get a sunburn; ultraviolent rays are carcinogenic; you start getting apoptosis in your skin as you start killing off cells that are potential tumor cells because DNA has been damaged.

You're exposed to somebody else's cigarette smoke or you smoke yourself and you constantly have apoptosis going on, program cell death going on in the airways because you're producing DNA damage constantly.

Those genes cause those cells with DNA damage to go down a death pathway. You never hear from them again. Thus preventing the development of cancer. Most of us don't get cancer. It's not easy to get cancer. But one in four, about 25 percent of the people get cancer. And the reason that we don't get cancer is because of these series of protective genes that we have.

But what if in this DNA is one or more of those

death pathway genes? Not in the right place; it's not going to work; makes it more likely that the cell is going to go on through the life cycle and develop into a tumor because it's passing those mistakes on. One mistake is not enough. Two is not enough. Three is probably not enough. Then it starts getting unknown as to just how many it takes. It's different for different people. Makes us a very complex issue. What's enough for one person, for a given individual? Let me finish up by talking about that a bit. This is my last slide.

So I'm going to just reiterate what I said about cancer and then I want you to understand what happened during those decades of latency. Why did it take so long for the person's exposure to cause the cancer decades later? That's what I want you to understand.

So here is a cell layer. These are epithelial cells; could be any kind of epithelial cells; could be mesothelioma cells lining the pleura; could be airway cells, could be any kind of cell. Just think of these as lining cells. Now the artist has a couple of lightning bolts and he says DNA damage. Of course I know lightning doesn't cause DNA damage, but this is -- this artist is under the direction of a Nobel laureate who discovered some of these growth control genes. He knows very well what's going on and he's talking about

DNA damage from something in the environment that comes in and causes this DNA damage in the target cell in the lining. Now we're talking about cancer, so it's the lining cell of the airway.

The artist knows very well that as a result of DNA damage, cells die. So here he's got one of the daughter cells going off. The DNA is all clumped up and the cell is going to go off and it's going to die. And you never hear anything about it again. That's what you hope for.

But we're talking about a cancer. So if we're talking about a cancer, that means that one of the daughter cells with an error must survive, and so he's got this daughter cell now surviving. You can see the chromosomes. And he also has a tumor. You see this says tumorigenesis or tumor growth. And he's got all this odd color that I'll explain in just a second.

The space between this first daughter cell and the development of the tumor, I need about 20 or 30 or 40 years in that space because I'm going to tell you what's going on in that space and time. The artist didn't help me by putting these together, but that's okay. Just envision that space.

So think about a single cell with DNA damage sitting on the surface of the airway. And it will sit like that for months looking and acting like a normal

airway lining cell. One gene, one error is not going to be enough to cause a cancer and it's not enough to effect the function of that cell. So it sits there looking and acting like a normal cell. As I say, it can sit like that there for months, but eventually it has to divide. So it divides: Two cells, four cells, eight cells, all with that same error.

Now, some of them die; some of them get hit again. Another fiber. Another cigarette. Whatever is causing the cancer. Hits that cell and you get a second error in a gene that controls cell growth. Now that cell has two errors and it sits there looking and acting like a normal mesothelioma -- I'm sorry, airway lining cell for months. And then it eventually has to divide: One cell, two cells, four cells, eight cells, sixteen cells, thirty-two. Some of them die. Some of them get hit again.

Now the field is spreading. The field of cells with errors is now spreading because you're getting more of them. There are patches in which you get several cells together with one or more errors. Not cancer. Now keep doing that. Go through that scenario with cells dividing in these various fields with varying numbers of errors. Do that for decades. Okay? And the lungs, the airways of people who smoke cigarettes over

their lifetime and get cancer, some of them have been tested over time and you can see these fields of errors, genetic errors. People who smoke and are exposed to asbestos have more errors in those fields. Eventually a single cell with sufficient errors for that person grows out and that's why the artist made this tumor a single color, because it came from a single cell. There are a lot of cells with errors and multiple errors, but only one grows out from a clone and forms the tumor that eventually brings that person to the clinic. That's why it takes so long.

THE WITNESS: That's all, Your Honor.

Q Dr. Brody --

THE COURT: You want him to look at the slide or --

MR. MCCOY: Yes. I want -- just a couple questions.

A Yeah.

Q So what you're saying is this yellow area is actually a single cell.

A Well, it's grown from a single cell. It originated from a single sell with a series of genetic errors.

Q And this represents the genetic errors that --

A Okay. So if you were to take the nucleus out of the cell or if you were to see how these chromesomes

were lined up, remember how I showed you the chromesomes lining up? If you were to look at these chromosomes or you were to take this cell out and and you were to sequence the DNA, sequence the genes of this person, you would see the errors.

Q So you would see multiple errors.

A You'd see multiple errors, and that's been done many times in numerous tumors from people exposed to cigarette smoke, people exposed to asbestos, people exposed to combinations of those.

Q Can one error cause a tumor?

A Highly unlikely. So we have a couple of cancers where we know a single error is enough. There are very few. I can give you an example. The BRAC1, B-R-A-C1a gene causes a very high likelihood of breast cancer in some Jewish sects. That's a single error that's likely to cause like 80 percent likelihood of getting a cancer. Very rare. But the answer is yes, there are some kind of GI cancers where a single gene is sufficient to cause cancer. But again, very rare.

Q Not usually from lung cancers.

A I don't think there is a lung cancer where a single gene is sufficient, single gene error is sufficient. I'm not familiar with that.

Q I'll let you take your seat. I have just a couple

more questions.

THE COURT: Sure. I'll turn the lights back on too.

MR. MCCOY: When I say I have a couple errors on this same topic -- couple questions on this same topic, Judge, and then I've got some more after that. But I'll finish this topic.

THE COURT: Sure. Well, let me ask for break purposes because it's been not quite 90 minutes with the jury. Is this as good a place as any to take a 15-minute break and finish the direct?

MR. MCCOY: Yeah. Almost. Let me finish with a few more questions.

THE COURT: You let me know when it's a good time and we'll break there.

MR. MCCOY: Thank you.

BY MR. MCCOY:

Q So when we get down to the number of errors to cause a lung cancer, how many does it take?

A I wish I could tell you.

Q Or does it depend on the person?

A Yeah, that's right. It depends on the person. There is no set number. It's a different -- between different rats and it's different between different people. And it's because we keep knocking out genes

with errors as the potential for tumor develops, but one keeps and several keep sneaking through and sending their cell lines out. I mean cancer is the most incipit form of natural selection. You know, Darwinian survival of the fittest. What happens is we start making cells that get killed by our own defenses and then one or more of these cells becomes immune, using that word correctly, because our immune system is very good at recognizing potential cancer cells. But it eventually reaches a point where they're not recognized by the immune system; they're not killed by those genes, protective genes I was telling you about. That's survival of the fittest. And that cell develops into a tumor.

Q We talk about lung cancer. Stay with that for a moment. What would be a typical number at which you might -- number of errors on a cell that might then cause an actual lung cancer? Give me a number.

A You know, that's very hard to answer because you can find tumors with anywhere from 5 to 15, sometimes even 20 separate errors, and the problem is you don't know which combination is the one that actually -- I mean, you know, people have been -- billions of dollars have been spent. There are spectacular investigators at the National Institutes of Health trying to sort out

what you're asking me. Which errors? How many? What does it take to form a tumor in a given individual? How do you stop it?

I mean we're studying right now with my colleagues at Tulane, we're studying what are called *cancer stem cells*. In other words, you can treat a cancer initially, knock out a lot of the tumor. It even looks like it's going away. But in fact, what remains is what's called a cancer stem cell, and years later that stem cell has been selected for and not is responsive to any treatments and it grows out again.

And there's nothing you can do about it. You can't treat that cancer stem cell. So that's just an example.

Q Can a single asbestos fiber cause an error?

A Yes. A single fiber does not cause the disease. Okay? A single fiber does not cause any asbestos-induced disease. But a single fiber can cause a genetic error. Sure it can.

Q Now if someone had like a cube of sugar size or I guess oftentimes it might be like one centimeter in your language, but something about that size like that, and you were to smash that up, how many fibers would be in that? Asbestos fibers.

A Well, it depends. I mean you can -- so that's a cc. A sugar cube size. A cubic centimeter is the way

we usually talk about it. Out in the background, out in the air if you walked outside here in Madison, I expect you to be able to find .00001, like a thousandth of one fiber. In other words, you'd have to have a thousand cubes before you could find a single fiber. That's what that means. So very few.

Sitting in a workplace with an insulator, an insulator -- have been recorded some of them working in mines and insulators, a thousand fibers in one cc.

MR. MOORE: (Stands)

THE COURT: I'm sorry. Do you want to object?

MR. MOORE: This is outside the scope of his report, Your Honor. He's not a industrial hygienist.

THE COURT: Sure. I haven't read the report. But Mr. McCoy, let's limit the testimony now to what's part of the revealed report.

BY MR. MCCOY:

Q What I wanted to do is basically just talk about the sugar cube. You've talked about the fiber and how those break apart; how many inside of that if you just broke that apart rather than talking about the background level of the air?

A Yeah. Well, as I say, you can -- that can vary depending upon the environment that you're in. And the animals that we expose asbestos to, we get about a

thousand fibers in one cubic centimeter. That's a heavy exposure. You can go down into hundreds of fibers or single fibers per cubic centimeter and those are all well above the background.

MR. MCCOY: That's all the questions I've got on this line, Judge, so I'll --

THE COURT: Okay. Fair enough. Well, Ladies and Gentlemen, why don't we take your 15-minute morning break and they we'll come back and continue.

(Jury excused from courtroom at 10:32 a.m.)

THE COURT: Doctor, certainly you don't have to remain on the stand. You get a break, too. But the usual rules apply. Because you're still on direct, you may not talk to anyone on your lawyer's team about your testimony during the break. Understood?

THE WITNESS: Thank you, Your Honor.

THE COURT: With that, you're free to leave the stand. Anything else then before you guys take your break? Mr. McCoy, about how much longer do you think on direct? Ballpark estimate.

MR. MCCOY: Roughly about a half hour.

THE COURT: Okay. Well, how roughly? 20 to 40? 30 to 60? Close to 30 minutes?

MR. MCCOY: Very close to 30.

THE COURT: Okay. Who's got the cross?

MR. MOORE: Me.

THE COURT: Mr. Moore, just a ballpark prediction from you.

MR. MOORE: 20, 25 minutes maybe.

THE COURT: Okay. So clearly we'll finish with Dr. Brody before the lunch break.

MR. MOORE: Yes, sir.

THE COURT: Okay. Fair enough. I don't have anything else. Mr. McCoy, anything else before you take your break?

MR. MCCOY: No, I don't have anything else right at this moment, Judge.

THE COURT: And that's all I'm asking. Mr. Moore, anything else at this point?

MR. MOORE: No, sir.

THE COURT: Okay. Then everyone can take their break.

(Recess 10:33-10:45 a.m.)

(Jury brought in courtroom at 10:45 a.m.)

THE COURT: Everyone please be seated. Ladies and Gentlemen, welcome back. Dr. Brody, just for the record, let me remind that you're still under oath.

Mr. McCoy, if you would continue, please.

MR. MCCOY: Thank you, Judge.

BY MR. MCCOY:

Q Dr. Brody, I don't want to recreate or reiterate everything that was on your slide show, but --
A That's a good idea.
Q -- is it possible then for a person to have more than one asbestos-related disease?
A Absolutely.
Q And I think the examples that you showed us on the one slide is somebody could have lung cancer, asbestosis, and pleural plaques?
A Oh, yeah. In fact, if somebody has the disease asbestosis, they're more likely to get a lung cancer. That means they had an exposure that can produce a lung cancer. If they have pleural plaque, they're more likely to have a lung cancer. Sure.
Q So how is it that it can happen that a person can get more than one of those conditions?
A Well, what happens is you have several different responses. So, if you -- you know, an easy thing to understand is if you scrape your skin away, you can get a scar. You also can get a melanoma from the sun. Those are different cells, different target cells, but they're two different diseases in skin that we're all familiar with.
Same concept in the lung. You can -- in fact that target cell for scarring, the fibroblast, by injuring

the surrounding cells, injuring the macrophages and the carpet cells, you injury those, you up-regulate the fibroblast, they make scar tissue. It's not cancer. But then at the same time, that same exposure exposes airway lining cells and you go through all the steps that I did to produce a cancer.

Q Is there any likelihood that someone would get asbestosis before the bilateral plaques or plaques before they get lung cancer or is there any sequencing order?

A No. So pick any order you'd like. In other words, what is the scenario. If a person is exposed to asbestos in an occupational setting and they have enough exposure to get asbestosis and a lung cancer, both of those diseases started very early in the process. As soon as the exposures start, you start producing genetic errors, you start activating the fibroblast to make scar tissue. They're both ongoing in the lung.

Now, which ones are going to bring the person to the clinic you don't know for decades. That's what I was telling you. The lung -- the asbestosis takes so long because we can deal with a lot of scar tissue in our lungs before it actually makes you short of breath. We have a lot of reserve.

I'm sitting here right now using about 20 percent

of my lung capacity. I get out and start running, I'm going to start using 50/60 percent of my lung capacity. So we have a lot of reserve. So as the scar tissue develops and you're preventing the movement of oxygen/carbon dioxide, you don't actually notice that you have the scarring going on in your lung until you start getting short of breath. Then you go to the doctor and he takes the x-rays and he gets your history and he finds out well, you've been exposed to a lot of asbestos and it looks like you're getting scarring.

Now, whether or not -- that may very well happen before the cancer shows up because the cancers usually develop near the end, closer to the end of the person's lifespan. That's what cancers typically do. It takes a long time to get past all the defense mechanisms. The scarring might show up earlier, but if you said well, the cancer showed up and then we found he had scarring, that certainly can happen. And say the same thing about pleural disease. Same concept. No set sequence.

Q When someone inhales asbestos fibers, you indicated sort of a process by which ultimately those fibers could become bound or DNA could become bound to those fibers; right?

A Right.

Q How long does that process actually take from the

time that someone inhales fibers or the range of time it takes?

A Yeah, that's very quickly. That's one of the first things that can happen. Because once the fiber gets into the cell and if it gets into the nucleus, as I showed you, within minutes of it getting into a dividing cell. So let's say here is an airway lining cell sitting there and a fiber lands on it and it gets picked up, but the cell is not dividing. Well, it can sit there for days or weeks and the cell doesn't really care if it has a couple of fibers in it.

But like I say, eventually it has to divide, and then the DNA is exposed. So, you know, if you want to count the time that the fiber sits there, it can sit there for months. But once the cell is dividing or if it lands on a dividing cell, almost immediately you can start getting genetic damage.

Q When you look at those -- the bound DNA to the asbestos fiber, what kind of magnification are you looking at?

A Well, this was a light microscope. A very good light microscope. You can start seeing it around a thousand times, 500 to a thousand times magnification. It's a lot easier to see with an electron microcope, but you can do it about a light microscope.

Q In terms of somebody going to the doctor's office and getting an x-ray, does that kind of binding of a single cell or the single cell and the fibers being bound with the DNA, does that show up on x-ray?

A Certainly not. Not even close. The x-ray -- x-ray is a very obtuse; not a fine tool. You need a lot of scarring in your lung before it shows up on an x-ray. X-ray is really casting a shadow. You know, you take an x-ray through the lung and most of the lung you can -- you know, very thin lines. All the walls and the carpet and all that is very thin. And the x-rays just go right through that.

The normal lung has a very clear shadow. So you start producing scarring caused by asbestos, you start seeing lines of shadows that are cast. And you have to have quite a bit in there actually before you can see it on an x-ray.

Q Latency period. What does that mean in terms of what we're talking about?

A The definition is time from first exposure until the time the person comes to the clinic. For all the asbestos diseases, it's decades. I explained to you why. I explained to you why cancer takes so long, because of the -- it takes a long time to get past all of our defenses and accumulate genetic errors.

Asbestosis takes a long time because you can produce a lot of scar tissue in the lung before you feel it. So the scarring really starts very soon after, but you really don't know it until enough of it accumulates.

The other thing that I really neglected to point out in all this is that lung cancer, all the cancers, asbestos-induced cancers, not only are caused by binding of DNA, but asbestos also generates what are called *oxygen radicals*. Now you may have heard it's a good idea to take antioxidants. I'm not telling you to do that. What I'm saying is that we have, as we walk around, we have a very careful balance of these very high energy compounds called *oxygen radicals*. They're naturally-occurring compounds. They occur as we metabolize. They occur when we get injury or inflammation. And we have a lot of naturally-occuring antioxidants that keep a very clear balance.

The reason we do that, that we have to have this balance, is because oxygen radicals damage DNA. That's been known for a long time. Oxygen radicals damage DNA. Asbestos, all the asbestos varieties generate oxygen radicals. That's one of the reasons, along with binding DNA, that they're such powerful carcinogens, cancer-causing agents. Cigarette smoke has a lot of oxygen generators, oxygen-radical generators. That's

the reason that cigarette smoke is a highly potent carcinogen.

Q You said something about latency period was the time from the inhalation until someone comes to the clinic.

A Right.

Q Comes to the clinic and what happens?

A Well, of course it depends on the disease; right? If the person is short of breath, he'll be dying -- he'll go through a series of pulmonary function tests and the doctor will determine whether or not he has an obstructive pattern like you get from cigarette smoking or a restrictive pattern. Obstructive means you can take in a deep breath, but then you're obstructed from exhaling and you're trapping carbon dioxide. That's typical of cigarette smoke. Or if you have your restrictive pattern which is caused by the scarring. I told you restrictive from taking a deep breath. So the doctor will determine that and he says if you have a restrictive disease, then you can go back to when the exposure started and you'll determine the latency.

Cancers, again, those won't be detected until the person actually shows up with a tumor, which is unfortunate because these tumors are typically life ending.

Q So latency period means from inhalation until actual diagnosis.
A Right.
Q In the meantime, that process you talked about is ongoing?
A Exactly.
Q Now when we talk about the inhaled fibers, do most of those stay in the lungs and cause the changes shown in the slides or --
A No. Actually most fibers that are inhaled are cleared from the lung. Ninety percent of what gets down into the lung, particularly in the airways, gets cleared out. And even that -- so it just shows you that a person who's exposed environmentally or in an occupational setting, you're exposed to so many trillions and billions of fibers, that even though you're clearing 90 percent of them, you still have enough to cause disease.
Q What about cigarette smoke? Is there a clearance for that also?
A Well, it depends on what you want to know about cigarette smoke. So cigarette smoke does a lot of things to clearance. It actually slows the clearance of asbestos. Cigarette smokers typically have more fibers in their lungs than nonsmokers. But the thing is that

once a person stops smoking, the likelihood of getting cancer from that cigarette use gets less every year. The risk goes down every year after that person stops smoking; never goes back to the nonsmoker, but goes down in a clearly measurable way so that if a person ends up with a cancer decades later and you look at the smoker history, you should know what that history is so you can understand whether or not it played a role in the development of the cancer. What was he exposed to? Was he exposed to cigarette smoke? Was he exposed to asbestos? Was he exposed to both? And for how long? Those help you understand what caused the disease.
Q Some of the clearance mechanisms that you mentioned for the fibers included the macrophages?
A Right.
Q And how is it that the asbestos fibers affect those macrophages? You showed us a picture where some of those have been --
A Sure.
Q Looked like they were beaten up or battered.
A Right. Exactly. So that's sort of a science in and of itself. I published a series of papers trying to understand that question. What is it about the fibers that causes the macrophages to deteriorate. A couple of different answers to that. One is that they're highly

charged.

So for example, chrysotile asbestos has a positive charge. If you look at the surface of cells like macrophages and epithelial cells, they have a negative charge. And just like happens in anything with positive charges, they attract, they bind, and they can cause damage.

We did a series of studies looking at the membranes of various kinds of cells like macrophages and found that there were distortions in the membrane. Various different cells are distorted by binding of the fibers, and these distortions in binding cause the cells to break down, leak, activate genes. There's a lot going on.

Q What other clearance mechanisms are there besides the macrophages?

A Well, you have the mucociliary escalator of course. So the escalator and then the macrophages are the primary clearance mechanisms once things get into the lung.

Q What's the escalator do?

A Well, everything that lands in the airways gets swept up, except of course for those fibers that are actually taken up by those cells. Those are the fibers that can interact with the DNA. So the fibers that get

into the airways are the ones that can interact with the DNA and cause lung cancers. Fibers that get down onto the carpet are the ones that, if they're not cleared -- and as I told you about 20 percent of those are taken right into the structure of the lung -- and those are the ones that are interacting and causing scarring.

Q Do some fibers break down and resolve?

A Yeah. And so there's another -- actually you just reminded me there's actually another clearance mechanism that we didn't talked about. It's called the *lymphatics*. You've probably heard of your lymph nodes. This is a clear fluid that flows in the lung. And when fibers like chrysotile, for example, break down in the lung, they can get transported to the pleura. And the predominant fiber type -- and they get transported in the lymph, lymph flow to the pleura. And that's why the predominant fiber typing in the lymph nodes and in the pleura is chrysotile.

As I said, all of the diseases are caused by all of the fibers. But chrysotile tends to predominate in the lymph nodes and in the pleura.

Q Do the clearance mechanisms remove all the inhaled asbestos fibers?

A No. Not even close.

Q Why not?

A Well, remember I said 90 percent. Okay? So if you're talking how many billions and trillions of fibers we're talking about getting inhaled by a person who's occupationally exposed? Take 10 percent of those, you know, you've got -- you still have billions of fibers. So those are the ones that get taken into the structure of the lung. And they'll never be clear. There's always clearance going on, but there's always some proportion.

You know, we published a series of papers looking at the lungs of people who died natural deaths or were killed in accidents and had a long history of occupational asbestos exposure. Some of them were 20, 30, 40 years past their exposures had ended and there's still trillions of fibers in their lungs because they get entrained into the connective tissue and into the walls of the air spaces, walls of the airways, and that's why they don't get clear.

Q If a particular fiber gets broken down and dissolved after it's penetrated through the lung, does that mean it has nothing to do with the errors that might result?

A Well, chrysotile is the only fiber that could actually break down. Crocidolite and amosite don't really break down over time. But chrysotile can get

into the lung. 95 percent of the world's use. So there is a lot of interest, and understandable, about chrysotile. Chrysotile can get into the lung, go to anywhere in the lung, cause genetic damage, cause scarring, and then be cleared. So the answer to your question is sure, that can happen. It happens over and over again.
Q I'll go back to your background briefly. Have you won any awards or gotten any awards or honors for your work in asbestos disease?
A I have.
Q Could you describe one or two for us?
A Well, I've had -- I had what's called the *Hatch Travel Award*, which means I'm given a nice sum and asked to speak before the environmental assembly at the American Thoracic Society. Theodore F. Hatch Award it's called.
I've been awarded a cash award by the National Institutes of Health. Not the grants. I mean separate from that.
I also was awarded a grant or a -- I was awarded a speaker's opportunity by the drug company called GlaxoSmithKlein. So they sponsored a trip to the university where I gave a talk to a group. Those are awards. But I get asked to speak at various places. I

consider that an award.
Q Including Madison?
A Including Madison, absolutely. I also was awarded -- I could say I was awarded the opportunity to sit in front of the U.S. Supreme Court. I couldn't address the court, but the very testimony you heard here today with my name is in the record when they were considering a case regarding asbestosis and lung cancer. So that was a -- I felt highly awarded by that opportunity.
Q When did you first start publishing in the field of asbestos disease?
A Well, I started looking, as I say, at the concepts in the 70s and started doing research in the late 70s, and published the first paper in 1980 in asbestos disease.
Q And you continued to publish after that?
A Yeah. My most recent paper is 2012. Absolutely.
Q These papers are published in what types of publications?
A Well, they're called *biomedical journals*. They are read by scientists in positions around the world; published in about probably 20 different journals over the decades.
Q Okay. You mentioned at the beginning some ongoing

research concerning cigarette smoke and asbestos.
A Right.
Q Had this been a field of research before the current research?
A Right. So for years we were, as you've heard already, asking various questions about how asbestos causes disease. One of the big issues related to asbestos exposure is what happens when people smoke and are exposed to cigarettes. There's a real synergy there. In other words, cigarette smoking causes -- gives you a very high risk of cancer. Asbestos exposure alone increases your risk of getting cancer, but not as much as cigarette smoking. But if you combine the two -- you can't just add it, you can't just add those risks, you have to multiply the risks. Now that means there's a synergy. So whatever it is that's causing the cancer in either of those carcinogens, there's something about the two together that multiplies the risk, and so that's enticing for a scientist to try and figure out what that is. They're both very complex carcinogens, but what are they doing?
So we're using animal models. We're using the cell studies that I showed you to try to understand that.
Q When you say the term *synergistic*, that's what you were talking about; right?

A   Yeah. It means multiplicative, yeah.
Q   How does that term synergistic apply to a person that's got both asbestos inhalation and some tobacco exposure?
A   Right. So the way that goes, what we understand now is that asbestos binds the carcinogens of cigarette smoke. And I've already showed you that asbestos gets into the DNA of cells. Now think of this as a very efficient way of introducing a potent carcinogen into the DNA. Asbestos is potent. Cigarette smoke is potent. But bind the carcinogens to asbestos and you have a very efficient delivery system into the DNA --
Q   Why --
A   -- of the target cell.
Q   Why does the binding of the cigarette carcinogen to the asbestos create a more efficient delivery mechanism?
A   Well, think about it for a second. Here if you have asbestos alone binding DNA, we know that that causes damage sufficient to produce cancer. Smoke cigarettes, introduce the highly charged oxygen radicals of cigarette smoke, that's enough to cause a cancer. Binding the two together creates a synergistic response.
      Now, how it does that? I mean that's exactly what we're trying to understand. I mean I say, you know, I'm keeping it in something we can all understand. This is

an efficient way to deliver the two carcinogens.

Now, I don't think you want me to get into the genetics of the inflammasome and the interleukins that are produced as a result of that because that's where the science is going and we're not there yet.

Q No sense in getting into research that hasn't been complete.

A I agree.

Q You talked about a hit I think was your word causing an error.

A You can call it that, a hit. That's good.

Q And then of course you mentioned that sometimes you were finding 15 or even 20 errors on someone who has cancer.

A Okay. Now that's not my research. Okay?

Q Okay.

A Scientists who are studying the genes that have been damaged in people and in animals with tumors have gone into those tumors and have found those numbers. That's not my work, that's other scientists.

Q Sometimes they could be less.

A Oh, sure.

Q My question is if there's a series of those hits, which is what you said it takes to cause the cancer --

A Right.

Q Okay. If there's a series of hits, can -- do all of those hits have to be an asbestos fiber or can some be an asbestos fiber and some -- one be a cigarette carcinogen?

A The answer is yes, and what else may be out there. In other words, you know, we're all exposed to various things over time.

Another thing is that once you start getting certain errors, we get what's called *genomic instability*. Our genes become more liable to injury when you already have some injury. So yes, asbestos can cause a series of injuries.

Cigarette smoke can cause a series of injuries. The combination obviously synergizes, and as that's occurring, other things can enter the picture. There are oxygen gases that we're exposed to. There may be other things in the environment that can cause these genetic errors.

Q Dr. Brody, is it the case that you normally, when you testify in courtrooms, you will be testifying for the -- on behalf of persons that have been injured from the asbestos?

A Typically, about 90 percent of the time in cases like this, yeah.

Q Have you testified though for the attorneys that

represent the companies in asbestos lawsuits?
A A number of times. I've testified for about ten different companies. Most recent was called *Plant Insulation Company*. That was in San Francisco last October.
Q And have you ever been asked and agreed to testify for an attorney representing Rapid-American?
A Absolutely. Sure I have. Yeah. Of course they asked me to give the exact same testimony that you've asked me to give here today.
Q About when was that?
A Well, so I told you the most recent one was just last October. Rapid-American asked me to testify for them in 1999.
Q And finally, your compensation is an on hourly rate?
A Right.
Q How much is your hourly rate?
A $550.
Q The amount that you charge would be the same regardless of whether you're testifying for a company or on behalf of an individual?
A Of course.
Q Okay.
MR. MCCOY: That's all the questions I've got.

Thank you. (11:15 a.m.)

THE WITNESS: You're welcome.

THE COURT: All right. Thank you, Mr. McCoy. Cross-exam.

MR. MOORE: Your Honor, if it please you, I'd like to sit at counsel table to ask my questions.

THE COURT: Yeah. That's fine. You bet.

MR. MOORE: Okay.

CROSS-EXAMINATION

BY MR. MOORE:

Q Is that okay with you, Dr. Brody?

A That's fine.

Q Okay. Hi. I'm Steve Moore. You are a molecular biologist basically; correct?

A In part, sure.

Q And a cellular biologist.

A Right.

Q You're not here to provide any testimony that any particular company or a particular product was a cause of any condition in Dr. or Mr. Bushmaker; correct?

A I didn't do that. That's right.

Q Okay. And you told us about your background and so forth, but you are not an expert in risk assessment or an expert in product warnings; correct?

A Correct.

Q You first started testifying in these types of cases back in 1989?

A I had one case in 1989 and a couple cases in the early 90s, yes.

Q And just to follow-up on a point that Mr. McCoy was making, really closer about 99 percent of your testimony has been on behalf of plaintiffs like Mr. Bushmaker in asbestos litigation; is that correct?

A That's fine.

Q Okay. How many times have you testified this year?

A Well, I get asked to do this several times -- could be two times a month, two or three times a month.

Q Right. How about -- have you even kept track of how many times you testified in a courtroom like this one in 2012?

A Well, just add it up. You know, it's about -- people seem to be interested in what I have to say, so, you know, I'm asked to do this -- could be two to three times a month. Sometimes it's once a month, sometimes it's three times a month.

Q So maybe 30 times a year.

A Approximately.

Q Okay. And last time I checked, you made over $200,000 a year testifying in cases like this for plaintiffs?

A Right.
Q You've received no information or case material specific to Mr. Bushmaker; is that correct?
A Correct. I was asked to make some assumptions regarding diagnosis and that sort of thing.
Q You haven't -- you don't have an independent opinion regarding those, do you?
A No.
Q Okay. In my opening statement, I provided some statistics to the jury about smoking and I wanted to just do a fact check on me. Okay?
A Okay.
Q Okay. Ninety percent of all lung cancers are caused by smoking; correct?
A Correct.
Q The majority of all lung cancers now occur in smokers that have quit; correct?
A True.
Q And unfortunately, more than a thousand people a day die from smoking-related illness; correct?
A True.
Q There are at least fifty cancer-causing agents, you call them *carcinogens*, in tobacco smoke; correct?
A It depends on whose list you look at. Some people can break them down into hundreds, but forty or fifty is

fine.
Q Okay. And that includes things like arsenic, formaldahyde, ammonia, benzene, hydrogen cyanide, and others; correct?
A That's right.
Q Okay. Is it true that one-half of all long-term smokers will die from a smoking-related illness?
A You know, I don't know about half, but -- I just don't know that number, but that sounds about right.
Q Okay. And there's no safe tobacco product, is there?
A Not that I know of.
Q You have alluded to this in your direct examination. You spoke of the asbestos-related condition, asbestosis, which is noncancerous; right?
A Right.
Q That's a restrictive lung disease; correct?
A Causes a restrictive lung disease.
Q Correct. And smoking tobacco causes a obstructive lung disease; correct?
A Right. I thought I explained that, yes.
Q Yes. And there's a clinical description of that called *emphysema*; correct?
A Right.
Q And emphysema is the -- we talked about the walls

of the -- the air sacs. They're also referred to as alveoli; correct?
A Alveoli, right.
Q And those look like grape clusters in the lungs; correct?
A That's fine.
Q Okay. And emphysema is the destruction of the walls of those air cells that you talked about in your direct testimony; correct?
A Right. It would be like breaking the wall down here. Instead of having individual courtrooms, you'd have one big courtroom and that decreases the surface area that you have for oxygen/carbon dioxide exchange.
Q Fair enough. Thank you, Doctor. The inhalation studies you've done with asbestos, they've all been done on rats and mice obviously; correct?
A Right.
Q We can't do those kind of tests on humans. That's unethical; correct?
A I agree.
Q And the express purpose for these inhalation studies that you've done is to actually create diseases in the rats and mice; correct?
A Of course.
Q And in your inhalation studies in the chamber that

you talked about, this 4 by 6 chamber, you expose these rats and mice to concentrations of 1,000 to 5,000 fibers per cc; right?

A As I described; right.

Q And that level, that concentration is 10,000 to 50,000 times what the current OSHA standard is for asbestos exposure; correct?

A Sure. But you just made the point. We want to -- we want to create a disease. We're not trying to protect the animals like OSHA does, we want to make sure they get a disease.

Q Exactly. That's my point. And the amount is 10 to 50,000 times the current OSHA level; correct?

A Yeah. That's a typical level that scientists across the world use in these animal models to produce a rapidly developing asbestos disease.

Q What we're getting back to here is that concept of dose or dose response; correct?

A Right.

Q And in opening statement, I talked to the jury, I'll take an aspirin a day to keep -- reduce the risk of stroke in me. Fair enough? That's good practice?

A Right.

Q But if I took 50 aspirins, it would probably be toxic to me.

A Right. Now, that's an analogy. But I don't think that's a good analogy. There are better analogies. Because when you take aspirin, a single aspirin like you should, it dissipates. And if you looked for aspirin in your system the next day, you wouldn't find it. But asbestos fibers, even at a low dose, are accumulating in the body over time. So I reject the aspirin use, but the concept of dose response of course is clear.
Q That's my point. It depends -- the toxicity of the substance depends on the dose; correct?
A Correct.
Q Okay. That's all I'm trying to ask you there.
A That's fine.
Q Okay. When you do these tests on rats and mice, you don't use an asbestos end product; correct?
A Correct.
Q A finished product. You use raw asbestos fiber; correct?
A Right.
Q And again, this is because you're more concerned about generating disease, not about dose; correct?
A Right. And it doesn't matter where the asbestos comes from. I mean the asbestos can come from a product; it can come from a mine. It doesn't matter where the asbestos comes from. Asbestos is asbestos and

it causes disease wherever it comes from.
Q But it does affect the concentration of exposure?
A No question.
Q Okay. And your studies -- would you describe your studies as cutting edge since 1974?
A I've heard that. I mean cutting edge studies are the only ones that get funded by the National Institutes of Health, yes.
Q No need to retread old ground; right?
A Right.
Q Okay. Your studies don't tell us what level of exposure is necessary to cause disease; correct?
A I've told that to the jury.
Q I'm just referring that in front of the jury here. And you're not trying obviously in these chambers, you're not trying to recreate or replicate any sort of working condition; is that correct?
A Well, we're not trying to, but I mean that's the concentration that was faced by miners and millers and some insulators decades ago.
Q And again, I hate to repeat what you talked about in your slide show, but I want to do more of a question-and-answer format. We all have some amount of asbestos in our lungs; correct?
A Right.

Q And obviously the vast, vast majority of us here are not going to develop any sort of asbestos-related disease because of these body defense mechanisms that you went through in great detail; correct?
A Right. There's no evidence that what we get from the background produces disease in anybody.
Q And I think in opening I said that -- this is fact check time -- I said that 90 percent of asbestos fibers are actually cleared from the lung. It's actually higher than that. It's probably closer to 95, 98 percent; is that correct?
A Yeah, it depends. I mean chrysotile certainly that's true. Crocidolite and amosite somewhat less.
Q Couple other small points. Aneuploid. I'm sure the jury remembers that word. You describe it as abnormal chromosome separation; correct?
A That's right.
Q And I think you alluded to this, but I mean any number of substances can cause aneuploid; correct?
A Sure.
Q Okay. And in particular, it can be caused by tobacco smoke.
A No question.
Q And we do know that it takes high doses of exposure to asbestos to create either lung cancer or asbestosis;

correct?
A Well, you know, what's high for one person is not high for another. So you don't know what it takes for a given individual. Cancers can be caused by less exposure typically than asbestosis, but if you have enough of an exposure which typically requires long-term occupational exposure, then you clearly produce conditions that will make lung cancer more likely.
Q I'm sure you probably can't recall every case you've provided testimony in, but there was a case called *John Markovich v. Bondex* in Dallas County where you provided some testimony. Do you recall that testimony? This was back in November 23 or 2003.
MR. MOORE: Your Honor, may I approach the witness?
THE COURT: For what purpose?
MR. MOORE: To impeach the last remark with prior testimony. Or can I use the ELMO?
THE COURT: Well, you don't have to show him the testimony to impeach him. Has Mr. McCoy got this transcript as well?
MR. MOORE: No. But I can certainly show it to him.
THE COURT: Mr. McCoy, why don't you two look at that.

(Pause at 11:25 a.m.)

MR. MOORE: Sir -- Mr. McCoy can read over my shoulder if that's okay.

MR. MCCOY: I don't need to, Judge.

THE COURT: And again, I don't want to micromanage this, but as long as we've got a foundation, as long as Dr. Brody agrees that's his testimony, then you don't have to have him read it to himself to impeach him.

MR. MOORE: No, I'm just going to ask him if this question was asked and if he gave this answer.

THE COURT: If he recalls.

MR. MOORE: Okay.

BY MR. MOORE:

Q In that trial, Dr. Brody, do you recall being asked the following question and giving the following answer:

"Question: We do know that it takes relatively speaking high doses of exposure to asbestos to create chrysotile asbestos to create either lung cancers or asbestosis; correct?

"Answer: That's correct."

Do you recall that testimony or that question and that answer?

A That's fine. I just told the jury just that. That impeaches me?

Q I didn't ask if it did or not, I just asked if I asked -- if you were asked that question and you provided that answer.
A If you read it, then sure.
Q Okay. Smoking has the effect of paralyzing the mucociliary escalator; correct?
A It can, yes.
Q Okay. And as a result of that, more asbestos fibers remain in the airways; correct?
A As I explained, that's right.
Q And in the airways, that's where the lung cancers arise; correct?
A It can.
Q How many genetic -- you don't -- I think you already testified to this. You don't know the number of genetic errors that are necessary to call lung cancers. It could be as few as five, it could be as many as twenty; correct?
A Sure.
Q And it depends on the individual person; correct?
A Exactly.
Q And there's no way of knowing which fibers caused the genetic errors that led to Mr. Bushmaker's cancer, is there?
A Well, you do in the sense that you can look back at

the exposure history. So you know what he was exposed to based on his history. Now you don't get to see the individual fiber binding the DNA.

Q That's my point.

A Well no, of course not. But you know when he was exposed and you know whenever he was exposed. That exposure introduces fibers to the system and any of those fibers can bind DNA and cause the disease.

Q Any or none of them; correct?

A Well, I wouldn't go with none of them because we know that asbestos is a carcinogen. If we know there's an exposure, we know the person has asbestosis, I don't see how you can dismiss the asbestos as causing some of the DNA damage. I don't know how you can do that.

Q Well, only because a lot of it is extracted from the body through the human defense mechanisms; correct?

A Well sure it is.

Q And many of the cells that are damaged die off through apoptosis?

A As I explained; correct.

Q So we don't know the specific fibers that cause the genetic errors; right?

A As I said, you don't get to see the individual fibers binding the DNA. But you don't -- but that doesn't mean you get to dismiss the asbestos exposure as

causing the disease.

Q We just don't know which ones cause those DNA hits that led to that particular cancer; correct?

A Yeah, I don't want -- you know, we're talking around a little bit because I agree you don't get to see the individual fibers binding DNA. So you look at what he was exposed to. Within that exposure were the fibers that bound the DNA.

Q Fair enough.

MR. MOORE: That's all I have. (11:28 a.m.)

THE WITNESS: You're welcome.

THE COURT: Done with cross?

MR. MOORE: Yes, sir.

THE COURT: Did you want to redirect?

MR. MCCOY: Just brief redirect.

REDIRECT EXAMINATION

BY MR. MCCOY:

Q Mr. Moore had read you something which -- I'm just go to borrow for one second -- about taking a high dose of exposure to create either lung cancer or asbestosis. Can you just give us some -- a little better quantification or something on what you mean when you talk about high dose?

A Sure. So in that trial apparently I said yes, it takes a high dose. And here I tried to qualify a little

bit and explain that it takes a relatively high, that is long-term occupational exposure or environmental exposure to produce asbestosis. That increases the risk of getting a lung cancer. So that means that that person also had long-term occupational exposure. But just how much again is different for different people. Some people can get a lung cancer from much more -- much brief -- a relatively brief exposure. So it's highly variable.

Q And we'll have additional people to talk about the occupational exposures, so I'm not going to add that to what you already covered in detail today.

MR. MCCOY: Thank you, Doctor.

THE COURT: So you're done with your redirect?

MR. MCCOY: Yes.

THE COURT: All right. Dr. Brody, you're done. You're free to go about your business. Thank you.

THE WITNESS: Thank you.

THE COURT: All right.

(Witness excused at 11:30 a.m.)

THE COURT: All right. And your next evidence, Mr. McCoy, is?

MR. MCCOY: Judge, we're going to go with one of the depositions to read in.

THE COURT: As you wish. Dr. Brody, have a

safe trip home.

THE WITNESS: Thank you, Your Honor.

THE COURT: All right. Who is your reader and who is your witness today?

MR. MCCOY: Today we're going to use James Hoey, who I should probably introduce as another one of the attorneys from my law firm.

THE COURT: Why don't you introduce him to the jury.

MR. MCCOY: Make sure everybody has got the right pages.

THE COURT: And you and Mr. Moore and Mr. Feldmann have consulted on this? Everyone knows what's in and what's out?

MR. MCCOY: Yes, Judge.

MR. MOORE: That's the plan, Your Honor.

THE COURT: I guess we'll find out.

MR. MCCOY: He'll object and call to my attention --

MR. MOORE: Best laid plan, Your Honor.

MR. MCCOY: -- and we'll fix it.

THE COURT: I'm sure you're going to do this, but let's lay the foundation for when, who, and so forth. And I'll remind the jury something we talked about Monday afternoon that this is evidence because it

is testimony at a sworn deposition. But of course Mr. Hoey is not the actual witness. So yes, listen to the words, but don't judge the demeanor. Okay?

MR. MCCOY: I'm just going to pull this podium up here.

THE COURT: As you wish.

MR. MOORE: Couple things, Your Honor. If Mr. McCoy could please, for the record, state what page of the transcript when he -- because I think it skips around a little bit.

THE COURT: You mean as he moves forward to alert you --

MR. MOORE: Yes, sir.

THE COURT: Sure. That's fair. Mr. McCoy, can you do that?

MR. MCCOY: Yes, I can do that.

THE COURT: Okay. Fair enough.

MR. MOORE: And I think that's it. I thought I had another issue, but I think --

THE COURT: Well, if it occurs to you again, stand up and tell us.

MR. MOORE: I will. Thanks, Judge.

MR. MCCOY: Jack of all trades. Moving the podium and reading the page numbers. This one won't be as long, Judge, as the other, but it's the second

deposition of Mr. Arthur Mueller, who was the witness we did before. It's a much shorter set of excerpts. This deposition was taken on February 11th, 1985. Spelling of Mr. Mueller's last name is M-u-e-l-l-e-r. Same person testifying as the other day.

MR. MOORE: I remember what it is. For the court reporter, for Lynette, did she -- do we want to transcribe this for the record or can she drop her hands?

THE COURT: Oh, no. That's actually a good pickup. As long as the parties can commit to me and Ms. Swenson right now that you can provide to us as an exhibit an accurate copy of the deposition that's being read, then we will not transcribe it at this time.

MR. MOORE: Okay. Thanks, Judge.

THE COURT: And let me tell the jury right now as I told you at the outset you don't get transcription of testimony. That would include this. So you will have to recall this. You will not get a copy of this transcript later.

So with that, let's begin.

MR. MCCOY: And Judge, for clarification I am reading both the questions and answers that were chosen by my law firm and the questions and answers that were chosen by Rapid-American's counsel.

THE COURT: Understood. Thank you.

MR. MCCOY: So doing both purposes here.

(Arthur Mueller depo excerpts read 11:33-11:57 a.m.)

THE COURT: Is that it?

MR. MCCOY: Just checking to make sure, Judge. Should have put flags on these ones at the end.

THE COURT: Take your time. That's fine.

MR. MCCOY: I'm not finding anything else.

THE COURT: So concludes the second testimony of Arthur P. Mueller. With that, we'll give you your usual hour for lunch and we'll resume at one o'clock. With that, you're excused.

(Jury excused from courtroom at 11:58 a.m.)

THE COURT: All right. Everyone please be seated. Let's just check before I let you guys go on your break. Mr. McCoy, any other issues that you need to front with the Court before we resume at one o'clock?

MR. MCCOY: I don't think so. I did want to say we're going to publish a few documents this afternoon.

THE COURT: Okay.

MR. MCCOY: Worker's comp claims. Subject to the pre-'60 rulings, I'll show them the worker's comp claims and I think a couple of exhibits at the deposition testimony from Mueller.

THE COURT: Okay. Well, let's break it out into smaller piece. So these are exhibits from the Mueller deposition or they're something else?

MR. MCCOY: I don't know that the worker's comp claims are from the Mueller deposition --

THE COURT: Okay.

MR. MCCOY: -- but they've been marked and testimony and these are the ones we provided.

THE COURT: Okay. Well, are they stipulated to foundation? I understand that -- well, let's just stop there. Is there any objection to the pre-'60 -- I'm talking to Mr. Moore and Mr. Feldmann now. Any objection to them being published, the pre-'60 ones?

MR. MCCOY: These are the ones attached, by the way, to the filing that we made --

THE COURT: No, I'm familiar with them, but now that we're at trial, I'm trying to avoid surprises. That's why we're talking before I let you guys go to lunch.

MR. MOORE: Yeah. Can we see the ones? I don't know which ones they are.

THE COURT: Sure.

MR. MOORE: Yeah, 'cuz I don't know which ones were allowed and which ones weren't allowed.

THE COURT: Well, pre-'60, anything up to the

point of last sale is in.

MR. MCCOY: I think the motion was 6-1-60 or something.

THE COURT: Well, I'm not focusing on a particular date, but yeah, we've got the cutoff. I guess the question then is a mechanical question from the Court. Sure, I don't mind if you publish them, but then in what fashion were you going to put the information in front of the jury? Were you simply going to --

MR. MCCOY: On the ELMO and just highlight certain portions of it. I mean they say Philip Carey Company and the disease.

THE COURT: Right, and that's exactly what I'm asking. When you say *highlight certain portions of it*, in what fashion? Were you going to read it aloud? Were you going to have someone else read it?

MR. MCCOY: I would read the highlighted portions.

THE COURT: Okay. Well then much like the Mueller deposition testimony, I'd like you to talk with opposing counsel about that and just find out if they want other parts read. And again, I don't know. I haven't looked specifically at these. But to the extent that they're going to be published and parts will be

read, I want both sides to have read those portions that they want read.

And I'm also assuming, subject to being disabused of this notion, that these will be published to the jury or sent back to the jury as exhibits at the close of the case. So that's not a decision that has to be made today. But again, as I told you at the outset, I expect the parties to keep track of those piles: Which ones have been admitted; which ones have been published; which ones do you expect to go back to the jury at the end. Okay?

MR. MCCOY: Um-hmm.

THE COURT: Mr. McCoy, anything else then on your agenda for the Court before we break for lunch?

MR. MCCOY: No. We'll go over the exhibits exactly how they're going to be highlighted as you said, Judge.

THE COURT: Sure.

MR. MCCOY: And the first witness after lunch will be Mr. Ferriter.

THE COURT: Is that the pipefitter?

MR. MCCOY: Yes.

THE COURT: Okay. Fair enough. Mr. Moore, Mr. Feldmann, any issues that you want to front with the Court before we break for lunch?

MR. MOORE: No, sir.

THE COURT: All right. Then you're free to go.

MR. MCCOY: Everything --

THE COURT: Or not.

MR. MCCOY: I just want to make clear everything that Mr. Ferriter is using we provided. It's all demonstrative to yesterday, so they can see exactly what it is.

THE COURT: Right. If I recall correctly, for reasons that are absolutely obscure to the Court, the question was make sure he puts a valve on it. I don't know why that's important. I'm sure it will become clear in retrospect. But in foresight, I have no idea why the valve is important, but I'm sure I will learn.

MR. MCCOY: He's actually not doing the drop cloth work. He's going to just be doing it through the ELMO and so forth.

THE COURT: I trust you guys to do whatever you want to do. I was actually looking forward to a hands-on demonstration, but if it's all going to be done through the ELMO, so be it.

With that, you're free to go to lunch. We'll resume at one. We're using the Court's clock for telling time.

(Noon recess 12:03-1:01 p.m.)

THE COURT: Let's go on the record, Counsel. Good afternoon. Mr. Moore, I understand that you want to talk to the Court before the jury came in?

MR. MOORE: Your Honor, just following up when we left about these claim forms --

THE COURT: Yes.

MR. MOORE: -- I think he's certainly entitled to publish these to the jury. If he does do any highlighting, I'm going to reserve the right to highlight at the same time. I think that's fair to me.

THE COURT: Which I think was what we all agreed at the break.

MR. MOORE: Okay. I can't -- I didn't recall that, but I'm --

THE COURT: Well, what I had hoped would happen was that that would actually occur during the break and so that it would be a done deal by the time you came back. But I'm not going to say it has to happen. I'd rather bring the jury in if we're ready to do that. But let's check. Mr. McCoy, with the claims forms, have you already determined -- Mr. McCoy?

MR. MCCOY: I'm listening.

THE COURT: Okay.

MR. MCCOY: I was looking for something else. I'm doing double tasking.

THE COURT: I know you're capable of that, but let's sort this one out. Have you decided yet which parts of the worker's comp claims forms you plan on reading to the jury?

MR. MCCOY: Yes, I have, Judge.

THE COURT: Have you shared that with Mr. Moore? And if you want to just show him --

MR. MCCOY: I was trying to get a yellow highlighter is what I was trying to do.

THE COURT: Do you want to borrow one of the Court's?

MR. MCCOY: I think I've got one here.

THE COURT: I've got one in my hand that might be quicker. Oh, you've got a whole pack.

MR. MCCOY: I forgot about this whole group here.

THE COURT: Well, but again, I don't want to keep the jury waiting too long.

MR. MCCOY: I can do this right now.

MR. MOORE: Your Honor, these do have settlement amounts in them?

THE COURT: And I presume you don't want the jury to see those as anchoring numbers.

MR. MOORE: That's correct, Your Honor.

THE COURT: Okay. Mr. McCoy, any problem with

blacking out the settlement amounts?

MR. MCCOY: No.

THE COURT: Do you have a black highlighter? I guess that would be a low lighter, wouldn't it?

MR. MOORE: A sharpy would be fine.

THE COURT: All right. Have you got one?

MR. MCCOY: Yeah.

THE COURT: Is there just the one or I mean are you guys all caught up then with all of them?

MR. MOORE: There are six total, I believe.

THE COURT: Have they all been properly marked and blacked out?

MR. MCCOY: We'll do them real quick, Judge. They'll just be done.

THE COURT: Okay. So we'll go off the record while you do that. Just give me the high sign when that's done, then we'll be ready for the jury. How does that sound?

MR. MCCOY: Yes. Very quick here.

(Pause at 1:05 p.m.)

THE COURT: Are we on the record?

MR. MOORE: Yes. It's been my experience working with sharpies that they -- I mean for this purpose, it'll be fine. What I'd like to do before the exhibits go back to the jury is to make sure that we

make an appropriate redaction.

THE COURT: Sure. That you photocopy them to make sure that the jury does not self-help and look up at the light and read the number through the blackout.

MR. MOORE: That's my concern, Your Honor.

THE COURT: I leave that in your bailiwick to implement, but you're entitled to that.

MR. MOORE: Thank you, Judge.

MR. MCCOY: Judge, I assume you'll explain the basic procedure on publishing; whatever you want to say on that.

THE COURT: I wasn't planning on saying anything, but I can. What --

MR. MCCOY: I was just going to introduce these as documents from the files of the Philip Carey Company and then I was going to state these are the parts that we're highlighting, calling to your attention right now --

THE COURT: Sure. Well, let me --

MR. MCCOY: That's about all I would say now. I think we might need some more background.

THE COURT: Well, that's fine, but let me suggest this: I don't know if you're starting with your pipefitter witness or if this is going to come later, but whenever you want to put the exhibits in --

MR. MCCOY: It will come now.

THE COURT: -- why don't you just announce to the Court that you'd like to offer your exhibits regarding worker's comp. I'll ask if there's any objection. The answer will be no. And at that point I'll admit them. At that point you may publish. It looks like you've got your computer guy or your IT guy there to help you publish, and then you can put them up on the screen and read whatever portions of them you wish, with the understanding that to the extent that Mr. McCoy has asked for additional highlighting, he's entitled to do that, too.

Now it's not clear to me from the process that you are now undertaking whether Mr. Moore has had an opportunity to look at them for highlighting purposes as opposed to blackout purposes. Mr. Moore.

MR. MOORE: I'm working on it right now, Your Honor. I'm almost there. For the record, there's the exhibit -- Hulette exhibit, whatever that one is. Clarence Hulette. This is an interesting case because he had an earlier claim that was disallowed and I don't think -- I think we should take that one out.

MR. MCCOY: It's not whether the claim is good or bad, Judge, it's simply that there was a claim made that put them on notice of this condition. Whether it's

a right or wrong claim is irrelevant here, it's only for the purposes of notice. We're not saying any of these people had this disease. We're just saying that --

MR. MOORE: If he'll stipulate to that, that's fantastic with me, Your Honor.

MR. MCCOY: That's exactly right. We would stipulate to that. We're not saying these people actually had this disease, it's just that they got this claim for that condition.

MR. MOORE: I'm fine with that stipulation, Your Honor.

THE COURT: Okay.

MR. MCCOY: I think that's basically the limiting instruction is what I'm reiterating.

THE COURT: Well, what the Court was focusing on was what I thought was a dispute about notice or danger and then we really are focusing on the post-1960 stuff, which is off the table now.

MR. MOORE: Right.

THE COURT: So pre-1960, I didn't know there was any limiting instruction requested at all. And again, I'm not remembering every issue that was raised in the 48 to 50 motions in limine. But Mr. Moore, is that your understanding with the pre-1960 claim forms?

MR. MOORE: Well --

MR. MCCOY: I recall some understanding about a limiting instruction on notice. I don't want to act like that was --

THE COURT: Okay. But that's what I'm trying to verify, whether anyone asked for it and whether the Court granted a limiting instruction on the pre-1960 claim forms as opposed to the post-1960.

MR. MOORE: There's two motions in limine. One was the worker's comp claim forms, which was 15, and then we had 23, which Your Honor sustained today.

THE COURT: Right.

MR. MOORE: And though there's some overlap, they're not identical. I understand claim forms come in for the purposes of notice. If Mr. McCoy is willing to stipulate that we're not offering these to prove that they had asbestosis or anything that's said in there, but merely to put us on notice, I'm okay with that.

THE COURT: Okay.

MR. MOORE: That our plant workers were -- that these claim forms were made.

THE COURT: Sure. Well, and how do you want to handle that? Mr. McCoy, do you want to simply indicate in the jury's presence that the parties have stipulated and then you say what the stipulation is and Mr. Moore agrees and then the Court accepts the stipulation?

MR. MCCOY: Um, that's fine.

THE COURT: Or do you want me to say it?

MR. MCCOY: That's fine. I just would prefer Your Honor give the stipulation. I can give the background; that these were employees of Philip Carey and that these were claims that they filed against the Philip Carey Company.

THE COURT: No, that's fine. But Mr. McCoy, you tell me first what you would like me to say. I want you to tell me your script for the stip; we'll run it past Mr. Moore, and then I am happy giving it. But I want you to tell me what you want me to say.

MR. MCCOY: My understanding -- my understanding of the stip is that these documents are being offered to prove that the Philip Carey Company had notice about the dangers of asbestos and that they are not being offered to prove that asbestos caused the condition of Mr. Bushmaker.

THE COURT: Okay. Mr. Moore. Did you catch that?

MR. MOORE: I did. I don't know if that's -- I feel like two ships in the night crossing here to some extent. I mean that would be an appropriate limiting instruction I would think under the circumstances; that these are only -- I'm having trouble here, Your Honor.

THE COURT: Well, let me ask you this: When is this coming up? Because we've now wasted ten minutes of jury time.

MR. MCCOY: I was going to do these right now. First thing.

THE COURT: Okay. Well, perhaps I wasn't clear. I expected this to be done over the lunch hour, even if that meant you guys only got 30 minutes for lunch. Now we're burning jury time. So, I want you guys to tell me what does the stipulation say that you want the Court to read?

MR. MOORE: Your Honor, I would propose the following: That the stipulation is that these are worker's comp claims made by plant employees of Philip Carey and they're not being -- I don't think we should focus on the notice; that they're not being offered to show that any Philip Carey product was defective.

MR. MCCOY: And I --

THE COURT: Let me suggest this, and again, I don't want to wrest control of this from either of you. But what if we simply were to say these worker's compensation claims are being offered simply to prove that Philip Carey Company received them and not to prove the truth of the matters asserted in them?

MR. MOORE: That's fine. I'd say, to modify it

slightly, not to prove, but as evidence of.

THE COURT: Well, as evidence -- these are documents that Philip Carey Company received. They are not being offered for the truth of the matters stated in them.

MR. MOORE: That's fair.

THE COURT: Mr. McCoy, is that okay with you? Do you want me to repeat it?

MR. MCCOY: (Nods head)

THE COURT: The parties have stipulated that these are documents that Philip Carey Company received. They are not being offered for the truth of the matter stated in them.

MR. MCCOY: I think -- let me just check. (Pause) That's fine.

THE COURT: You got it. All right. Are we ready for the jury?

MR. MCCOY: That's something then Your Honor will read.

THE COURT: Yes. You just give me the high sign and we'll read it.

MR. HANBURY: Your Honor, if it's all right, we'll take the screen down. Just in case we need a side bar, it won't be in the way.

THE COURT: Can you do that in 30 to 60

seconds?

MR. HANBURY: We can, Your Honor.

THE COURT: That's why you have young healthy help; right?

(Jury brought in courtroom at 1:15 p.m.)

THE COURT: All right. Everyone please be seated. Ladies and Gentlemen, welcome back. I apologize if we didn't start right at one, but the lawyers and I had a couple of matters to clear up.

With that, let's continue. Mr. McCoy, what have you got next?

MR. MCCOY: Next, Judge, we are offering some documents from the files of the Philip Carey Company.

THE COURT: Okay. And these are worker's compensation claims?

MR. MCCOY: Yes. These are worker's compensation claims made by employees of the Philip Carey Company.

THE COURT: All right. Any objection to admission?

MR. MOORE: No, sir.

THE COURT: All right. They're in. Would you like me to provide the stipulation to the jury at this time?

MR. MCCOY: Sure. That's fine.

MR. MOORE: Your Honor, I reserve my objections previously stated prior to trial.

THE COURT: Understood.

MR. MOORE: With that --

THE COURT: Understood. We'll talk about that at the end of the day. Ladies and Gentlemen, what the parties have agreed to is that the documents that Mr. McCoy is going to publish to you on what we call the ELMO, it's the acronym for our evidence presentation system, are documents that the Philip Carey Company did receive. However, they are not being offered and you should not accept them for the truth of any of the matters asserted in them. Okay?

So with that, Mr. McCoy, you may publish, and to the extent that you'd like to highlight any portions of those, you may.

MR. MCCOY: Okay. These are prehighlighted, and the first one -- I may have to get the sizing on this right. This is plaintiff's -- so this is going to be Exhibit No. 529. Does this automatically focus? Yeah, got Dr. Brody's electronic microscope. That's a good idea.

So this is Exhibit No. 529. And we'll go section by section on this. This is a claim by Albert S. Johnson. Deceased. Age is cut off, but he's 50

something. His employer as it shows there is the Philip Carey Manufacturing Company. Lockland. He died on March 24th. That would be 1949. Quit February 4th, 1949. The Nature of the Injury: asbestosis and heart failure due to it. Worked as a utility man in insulation division and also as a helper on Number 3 corrugator machine used to make corrugated paper.

And additional highlighting in Remark section is utility man. One other item on here. Date received: July 1, 1949.

Anything else I need to add, Mr. Moore?

MR. MOORE: No.

MR. MCCOY: That's the highlighting that was done by both parties.

THE COURT: Understood. Fair enough.

MR. MCCOY: Next is Exhibit No. 528. I assume this is clear enough to everybody. My eyes ain't perfect, so I figure if I can do it, most people can. And this name was cut off because it's -- let me just show -- the document itself is cut off a little. The piece of paper is cut off. That's why it looks this way.

So this was a claim by somebody. Deceased. And it's again Philip Carey Manufacturing Company would be the employer box. Lockland. It says this person quit

12-22-51. Died 9-14-52. And in the Nature of the Injury box, it says, what we can read, something about -- looks like enlarged heart caused from bad lungs. Fibrosis. And then --

MR. MOORE: Your Honor, I believe that says fibracosis.

MR. MCCOY: My mistake. Fibracosis.

THE COURT: It speaks for itself.

MR. MCCOY: Right. Okay. Of asbestos fiber from -- I can't read exactly the rest on that word, but looks like storage to conveyer. Opening and emptying bags. And then something into conveyer. And then additional, it says: Date received 1-20-53. And then down at the bottom highlighted it says: Asbestos beaterman helper.

Everybody read that? Okay. That was Exhibit 528.

And then so this is Exhibit No. 522. I don't think I'd make it in an eye doctor's office. Okay. This is a claim by Herbert I. Gooch. Deceased. Age 60 something. His employer was the Philip Carey Manufacturing Company. Cincinnati is listed for city. Date of injury is -- says 1955. Looks like it's scratched out or there's a line through that. April 1955 on it. He quit 4-6-55 and also 10-17-58. Says over here he returned 1-3-56 and died 8-25-60.

Nature of injury: Silicosis. Lungs. Worked in area where asbestos fiber and cement and silicate flour was -- were taken from bins, weighed and dumped into mixer. And then down at the bottom it notes that he was a shingle straightener. And that also the date received on this claim is 1-29-59. And that was 522.

Then Exhibit 523. This is a claim by Henry N. Hoerst. Age 59. Again employer is Philip Carey Manufacturing Company. Lockland. The date of injury, it's got something written on there about -- circled 1960, but it's kind of written over it. And it says -- next part it looks -- says here something about 1955, the date of injury box. Quit 12 -- 6-12-57 and 11-15 -- can't read the year that it says there.

Nature of injury: Asbestosis. Dumping asbestos fiber onto conveyer from burlap bags. And worked in asbestos beater room from 6-1936 until 2-10-1958. Date received is December 24, 1958.

And that's all on that document. That was 523.

Then we have Exhibit No. 521. The name of this claimant was Clarence A. Hulette. Deceased. Age 61. Employer: Philip Carey Manufacturing Company. City: Lockland. Date of injury: 1958. Quit: 4-13-59. Nature of injury: Asbestosis. Lungs and throat. Exposed to dust in beater room caused by asbestos which

was put in beaters. Date received: July 8th, 1958. And in Remarks it says: Janitor. That was 521.

Finally, last one, Exhibit 524. This was a claim by Essie Jackson. Employer: Philip Carey Manufacturing Company. Lockland the city. Date of injury says 1960. Says: Quit 9-6-60. And then down at the bottom in the Remarks it says Note: The company had no knowledge of this until it received claim number card. See correspondence. That was 524 exhibit.

THE COURT: I'm sorry, what date was that one received?

MR. MCCOY: It doesn't have a specific statement about received. All it says is date received. Copied (see note).

THE COURT: Thank you.

MR. MCCOY: That's our documents for this part of the presentation.

THE COURT: And those have been accepted into evidence.

MR. MCCOY: Thank you.

THE COURT: All right. Mr. McCoy, who is your next witness, please?

MR. MCCOY: The next witness is going to be taken care of by Kevin Hanbury, and I'll let him go ahead and do the introduction.

THE COURT: Very good. Mr. Hanbury.

MR. HANBURY: Thank you, Your Honor. Plaintiff would call Mr. Joseph Ferriter.

THE COURT: All right. Mr. Ferriter. Sir, if you'll please approach the court reporter, who will administer the oath.

JOSEPH FERRITER, PLAINTIFF'S WITNESS, SWORN,

DIRECT EXAMINATION

BY MR. HANBURY:

Q Good afternoon, Mr. Ferriter.

A Good afternoon.

Q Sir, could you please state your full name and tell us where you live.

A Joseph Ferriter. I live at 16354 Paxton in Tinley Park, Illinois.

Q And how long have you lived there, sir?

A Twenty-three years.

Q And are you currently retired?

A Yes, sir.

Q What did you do before you retired?

A I was a pipefitter.

Q And what year did you begin working as a pipefitter?

A 1953 I started my apprenticeship.

Q And how long did your apprenticeship continue after

1953?
A Five-year apprenticeship. Until 1958.
Q And what happened in 1958?
A 1958 I became a journeyman pipefitter.
Q Can you explain for the jury what a journeyman is?
A A journeyman pipefitter is a person that installs piping in oil refineries, powerhouses, nuclear powerhouses, fossil fuel powerhouses, industrial areas, hospitals; just about any place, even residential piping at times.
Q And did you begin working as a journeyman pipefitter in 1958 or did something else intervene?
A I was drafted in the Army in October 1958; served two years in the Army in the armored infantry.
Q Where did you serve in the armored infantry?
A I was stationed in Germany for two years. Eighteen months in Germany.
Q Did you return to the United States in 1960?
A That's correct.
Q And I assume began working as a pipefitter at that point?
A Yes. I went back to work as a pipefitter.
Q Are you a member of a union?
A Yes. Local 597 out of Chicago.
Q And what geographic area does that Local cover?

A Probably the easiest way to explain that is about 50 miles from the center of Chicago out from the Wisconsin border, all the way down into Illinois, around into Indiana, up to the Michigan state line.
Q When you worked with other pipefitters, did you largely work with other pipefitters from that local or did you work with pipefitters from other areas?
A Ninety percent of the time we worked for the pipefitters in the local area, Local 597.
Q And in your work as a pipefitter, did you work with workers in other trades?
A Yes. We were always mixed in with other trades, yes.
Q Could you give us some examples?
A Well, we worked with carpenters, bricklayers, electricians, iron workers, boilermakers, pipe coverers, tile men putting in floors, cement finishers. You name it, we worked with them.
Q Could you explain for us what a thermal piping system is?
A A thermal piping system is anything that has a heat source to it and transfers heat to something else. It would be just like if you have a hot water system in your house, you have a boiler downstairs, the piping takes the hot water to your radiator, which gives you

heat to warm your house. That's more or less a thermal system.

Q And could you describe for us what pipefitters do in industrial settings in terms of working with thermal piping systems?

A Well, it depends upon what the architect or the engineer wants you to do. They're all basically the same kind of systems. It's just that maybe the engineering outfit or the architect might have some added little Christmas tree stuff he wants to put on there. But basically they're all the same. It's piping that runs from point A to point B with maybe takeoffs to different equipment along the line.

Q And can you tell us what are some examples of industrial or large commercial facilities where you worked as a pipefitter on thermal insulation systems?

A The Thompson Center in Chicago is a good one. The Taylor Homes, which was a big housing subdivision that stretched from 55th Street in Chicago all the way to 35th Street, very big expansion job there. It was 18-story buildings. It was, I believe, 30 buildings there all together. And they were all heated with hot water.

Q Did you ever work in what we call a *powerhouse* or --

A I worked in nuclear powerhouses and fossil fuel powerhouses.
Q Can you tell us where?
A Yes. I worked at Braidwood Nuclear Powerhouse. I worked at Dresdin Nuclear Powerhouse. I worked at a place called -- let me think for a minute -- it's in Indiana. That was one of the fossil fuel places I worked at. I worked at Joliet Fossil Fuel Powerhouse and Stateline.
Q Have you ever worked in a paper processing plant?
A Not in a processing plant per se. I guess you would call it a processing plant. It was where Xerox used to make paper for copy machines. They had a coating they used to have to put on the paper. The paper came in in rolls and all they did is put some kind of a film or something on it so they could use it in copy machines. I worked in that place for awhile.
Q And what type of work did you do in that facility?
A We ran some high temperature hot water lines for that particular system and we ran the heating and air conditioning lines in the place.
Q Were you ever a supervisor as a pipefitter?
A Yes, sir. Most of my life.
Q Give us a time frame, if you would.
A Probably about -- after I got out of my

apprenticeships, 40 of the last 45 years I was a superintendent or supervision of some type.

Q Could you describe for us some of the large jobs that you supervised?

A Probably the largest one I supervised was Mobil Refinery in Joliet, Illinois. I had 1,300 pipefitters working for me. Braidwood Powerhouse we had about 650 people there. Union Oil in Lemont, Illinois I had about 200 people working there. Couple of the fossil fuel powerhouses usually range from 50 to maybe 100 men. A couple of the chemical plants I worked at we ranged anywhere from 300 to maybe 600 people. It depended upon the size of the project and how much they wanted done.

Q Now do the methods and techniques by -- used by pipefitters working on thermal insulation piping, do they vary from region to region or are they uniform?

A Basically piping is the same no matter where you do it. The scenery might be a little different, the climate might be a little different, but the work is all the same. It's a generic program of, like I say, the architect or engineer might have you put some extra parts or pieces in or might have you change different pieces, but it's all basically the same.

Q And how do you know that?

A From experience over the years from working in many

different places. A hot water system is a hot water system. They all work the same. Steam system. Steam system. They work the same.
Q And would that be true in a powerhouse versus a paper mill versus a factory?
A Yes, sir. There might be different pressures or different temperatures, but basically the basic method is all the same.
Q Are thermal piping systems insulated?
A Yes, sir.
Q And what trade does that work?
A The pipe coverers.
Q Have you observed the work of pipe coverers, insulators on thermal piping systems?
A Yes. On many jobs.
Q And how was it that you observed that work?
A They usually work in a very close proximity to us, and a lot of times it was my job to coordinate when the pipe coverer could come in and start installing pipe covering on the pipe we installed. So I worked pretty closely with them a lot of times.
Q By the way, have you ever worked in a supervisory role where the insulators were working directly under you?
A A couple of jobs they were, but the majority of

jobs the insulators are usually a separate entity. They're hired a lot of times direct by the contractor or possibly by the owner themselves.

Q Now when you're working as a supervisor installing piping of thermal insulation systems, are you required to sign off on that work when the work is complete?

A When the work is complete and all the testing is done and everybody is satisfied and signed off all the testing, it was my job to make sure that all this paperwork from this testing of the test runs we had done on the stuff and everything else was handed over to the general contractor. And he in turn, with me, gave it to the owner or the appropriate people it should be given to.

Q Now have you worked in industrial settings on high temperature water lines?

A Yes, sir.

Q And can you tell us where?

A The main place that I worked on where it was really high temperature hot water was the Braidwood Nuclear Powerhouse, and that's called a *boiling water reactor* in that place. What they do -- should I explain about that system there?

Q Well, first of all let me ask you this: When we speak of high temperature water lines, are we talking

about water lines with temperatures of at least 400 degrees?

A Yes.

Q Okay. And you said you worked on one at the Braidwood Nuclear Facility, and I think you told us you worked on one at the Xerox facility as well?

A Xerox facility and also Taylor Homes, which was a big housing project.

Q Could you explain for us what a high temperature water system is?

A All right. Water in its ambient state is -- boils at 212 degrees. That would be just like putting a pan on your stove, turn the fire on underneath, you see this little steam coming off the top. That's the hottest that water will get is 212 degrees.

Now, if you put a cover on that pot and seal it, as you heat that water up it will expand and it will cause more pressure. The more pressure you put on that water, the higher the boiling temperature. So if you take water that's under, say, 100 pounds pressure or 125 pounds pressure, the temperature that that water will boil at is 338 degrees because raising the temperature or raising the pressure raises the temperature, the water boils. So the higher the pressure, the higher the boiling temperature. That's why they call it a high

temperature hot water system, because it is above 212 degrees.

Q And sir, did you prepare a diagram for us of a --

A Yes, I did.

Q Sir, can you explain the diagram for us?

A Yes, sir. On the upper right-hand corner right here, you'll see a thing called the *accumulator tank*. That's where everything starts. That's where the main water source is and the whole bit.

Let me clear all that. From there it goes down to the pump down below here. This thing is not working too good over here. That pump boosts that pressure up to the pressure -- desired pressure that they want. Whether it be 200 pounds, 300 pounds, that pump is built to pump that water to that pressure.

From there, it will go to the boiler. That boiler is sized or engineered to take that water and bring it up to the temperature that you desire, whether it be 450 degrees, 500 degrees, or whatever the temperature might be.

From there, it goes out to the users, and what you call the users are anything that will transfer heat: A radiator in your house; a what they call a heat exchanger; it could be a process of some kind where you have to heat certain things or cool them off; whatever

will help. And then that system goes back and starts all over again. It's a continual system. It's one circle. It's closed. It's combined. It's one combined system in itself.

Q Okay. And you have a couple other more focused in diagrams. But before we talk about those, can you tell us what function does this system serve? What does it do?

A Well, usually engineering tells you that you might have to have a process that works correctly under, say, 450 degrees. They might want to take and maybe extract a certain kind of fuel off of crude oil. They have to bring this up to that temperature to extract something off. Something else might be extracted at 300 degrees. These systems are made with control valves to control this. The pump will control the temperature or the pressures that you want. The boiler controls the temperatures you want, and those are all set by what the users need.

Q And what purpose does a high temperature water system serve in, say, for instance, the Xerox paper?

A Okay. On the Xerox paper deal, they had to bring this paper in between these two radiators, I guess you would call them, and it had to be a certain temperature to apply this covering on top of it. And as soon as it

come out of there, it had to be cooled and then that Xerox paper is used in the Xerox machine. It's a -- I guess chemical engineers probably come up with this design or whatever, and they just transferred it out to us and tell us what to do with it.

Q Okay. And have you done anything to confirm whether the high temperature water system at the Consoweld plant where Mr. Bushmaker worked is of a sort that you've just described for us?

A Yeah. Me and Mr. Bushmaker sat down the other night. We kind of discussed our --

MR. FELDMANN: Objection, Your Honor.

MR. MOORE: Your Honor, this is outside the scope of his report.

THE COURT: I'm not going to forbid him from confirming what the Consoweld plant says.

MR. MOORE: Very well.

THE COURT: So let's keep going.

MR. HANBURY: Thanks, Your Honor.

THE WITNESS: We kind of discussed the kind of work that we had done at different places and the work that Mr. Bushmaker had confirmed that he had done at the plant there is very similar to the work that I've done in a lot of different places. Just like I said before, pipefitting is pipefitting wherever we go, just a little

different scenery. Maybe little different atmosphere. But that's about it.

BY MR. HANBURY:

Q Thank you. And Mr. Ferriter, could you tell us what this diagram depicts?

A Okay. This depicts a expansion joint. And what expansion joints are used for is, like I told you before, when you cover this pot of water and you keep heating it up, the pressure keeps rising. The temperature keeps rising. Now with anything that you heat up, it expands. When it cools off, it contracts. That's normal with anything in nature, anything in the world.

Basically on piping, water is one of the most, I guess you would say, volatile systems when you heat it up because water in this piping, if you take a 100 foot of pipe and you heat that water in that pipe 100 degrees, that pipe will grow one inch. So for every 100 degrees you grow, every 100 foot of pipe will grow an additional inch.

So when you have one of these systems, you have to take and you have to anchor it in certain places so it cannot move. It can't move any way at all. It's stationary. It's tied into the steel of the building or the concrete or whatever and then when this water heats

up, it pushes in toward this loop. What you see in the center. If you see the dotted lines in there, that's where that piping will be when that system is red hot and operating at the temperatures it should be. When it cools back off, it'll go back to its natural position.
Q So if I'm understanding you right, these pipes move.
A They move quite a bit, yes.
Q And sir, could you tell us what this diagram shows?
A Okay. This diagram here shows our main going by and shows typical drops going to all the users I had talked about earlier. Now these users are mainly connected with hoses and each one of these hoses has allowed the machinery and the piping to move because of this hot water always constantly moving and causing problems if it was stiff.
If you notice, there's an anchor down in the corner, down in the left-hand corner. That's to hold that header stiff so this piping up in here can move with the motion of the pipe moving back and forth. The equipment can't move because it's got a flexible hose with it. So all of these little parts that you see contain a big part of this hot water system, because if you tried to tie this down basically in one place, you would have a lot of broken parts by the time you got

through just heating this thing up.
Q And I see the words here two equipment type? What kind of a --
A That's typical. T-y-p is typical.
Q Oh, typical.
A So that one drop might be maybe a dozen machines, maybe only two or three machines, but it might fit a dozen machines. That's why I put typical down.
Q And in a paper processing plant, what kind of machines would we be talking about?
MR. FELDMANN: Object to the form. Foundation.
THE COURT: I'll let him answer if he knows.
THE WITNESS: It's most probably a heat exchanger type of some kind that will transfer heat from one to another, just like a radiator in your house.
BY MR. HANBURY:
Q Finally, Mr. Ferriter, just one more. Can you tell us what this diagram depicts?
A This is your pump assembly, and what you've got, this is the inlet of the pump, and always on the inlet side of your pump you have a shut-off valve, which is either open or closed.
Next to it you have a strainer. And what the strainer does is catches any foreign objects that might be in the water. Maybe a piece of slag that broke off

from the welding site; maybe somebody dropped a nut inside the pipe before they closed it up. Could be any little thing. Maybe some scale from the inside of the pipe.

Next you have what they call a flexible connection in there and that flexible connection eliminates vibration between the piping system and vibration that the pump would be causing.

Now on the discharge side, the other side, we have something very similar. We have the flexible connection again to keep from transferring any vibration to the piping system. We have a check valve, which is nothing but a gate. It sits in the water and it swings back and forth. Now if that pump stops, that gate closes and water cannot rush back in to injure the impellers in that pump. Then you have a shut-off valve again, which is either open or closed, which they use for maintenance to maybe remove the pump or work on the pump.

Q Mr. Ferriter, have you watched a video by the United States Department of Education called *Covering Hot and Cold Pipes*?

A Yes, sir, I have.

Q And does that accurately depict the manner in which water lines operating at, say, 400 -- in the 400 degree range were insulated during the 1950s?

A Yes.
Q And would that be true of land-based industrial facilities as well as ships?
A Yes. Any place a hot water system would be.
MR. HANBURY: Your Honor, with your permission we'd like to show that video to the jury. We've -- defense counsel has reviewed it and has no objection.
MR. MOORE: That's correct.
THE COURT: All right. Let's play it.
MR. HANBURY: Thanks, Your Honor.
MR. MOORE: This is from the 1950s?
MR. HANBURY: 1945 actually.
(Video played 1:52-2:13 p.m.)
MR. HANBURY: May I continue, Your Honor?
THE COURT: You may.
MR. HANBURY: Thank you.
BY MR. HANBURY:
Q Mr. Ferriter, I've got what I think is a short length of ten-inch pipe?
A No. That's six inch.
Q Six inch. Okay. Is this within the range of what you would find on a high temperature or water line in terms of diameter of the pipe?
A Yes, it would be. Yes.
Q What range do you typically see?

A It can range from three or four inch all the way up to 24 inch, maybe 36 inch. There's 24 inch in a lot of the boiling water powerhouses. A lot of small factories and stuff might have four, six inch, maybe eight inch.

Q And the video we just watched showed the application of pipe insulation, in your work as a pipefitter would you have occasion to remove piping insulation?

A Yes, we would.

Q Under what circumstances?

A Under possibly a leak. Or maybe if we wanted to get in to inspect something to make sure that we don't have a problem or maybe remove something like a control valve or maybe a specialty that's in the line that might have to be replaced or repaired.

Q I'd like to refer back, if we could, to your -- to the first diagram we looked at of a high temperature piping system. And could you identify for us what components of that system would have either block or cement insulation on them?

A Starting up by the accumulator, the RV on top, which is a relief valve which is a safety. The gate valve that's coming out of the bottom of the accumulator, that would probably have block insulation. Coming down to the pump, all the different fittings or I

should say valve and valve assemblies going into the pump section and the pump discharge would have all block insulation on them. Plus most probably the working part of the pump, not the driver, not the electric motor, but the driver into the pump would be insulated with block.

Going over to the boiler, you would have the valving going in and out of the boiler again would be blocked. Of course the whole boiler would be done with block insulation. The RV, which on a boiler is usually two or three of them, would be done with block insulation on top.

Going from the boiler up to the users now, all the valves and valve assemblies, the same way would be done with blocking. All the different users, depending upon what they were, if they were a heat exchanger, they'd probably be covered. If they were a radiator, that meant to disperse heat to a living area or something like that, they probably wouldn't be covered.

Q Now what purpose does the block and cement insulation serve?

A Well, first of all to control, or I should say conserve the temperature that's inside the piping because if it's a very high temperature and it's exposed to 70 degree air, it's going to take a lot of heat away from it. So you want to conserve as much heat in there

as you want.

If it's in a place where people might be working, it could be used as a personnel protection so you don't get burned in case you accidentally touch the piping.

Q How thick is the block insulation?

A It could vary depending upon the temperature in the piping. It could be a half inch thick, it could be three or four inches thick, maybe even six inches depending upon the process and the heat in the system.

Q What is a shutdown?

A Shutdown is normally when a factory or refinery or almost any place, they usually do a shutdown once a year to check their equipment to make sure that everything is working right. If they've had a problem along the year with a certain part of a system, it's a time when they go in and make repairs. They're usually scheduled for the economics part of the factory, plus for safety and want to make sure that everything keeps running properly.

Q And in terms of maintenance on shutdowns, is there maintenance done on the high temperature water system?

A That is one of the first systems they hit and usually the first part of that system that they do is the little RV valves that you see on top. They're not so little, they're pretty good size. Any place you have

an RV on a high temperature hot water line, you have many of them because they're a protection for that system. If something goes wrong, if something gets overheated, those valves relieve to the outside atmosphere to eliminate the danger or possibly catastrophe that could happen.

So the first thing that happens is all the RVs are taken off. They're sent out to an independent test lab where they test them to make sure that they are operating right; they're set at the right proper temperature, the right proper pressure, and then they are tagged and sent back in to the factory or wherever you're at.

Q And backing up just a minute, referring back to the diagram, I asked you where block insulation would be found on these various components. Where would cement insulation be found?

A Cement insulation would be found on any place where the block insulation would be because you have to seal up all the cracks because these -- most of these configurations that they are putting this block insulation on is rounded. So you're going to have separations in between the blocks that you put on here. That's where you use the mud to take and seal this all up.

Q So basically if there's block insulation, there's going to be some cement insulation.
A That is correct.
Q So in order to perform this testing on the component of the high temperature water system, are you removing the insulation?
A Yes. We have to remove the insulation. When we do a shutdown or go in to rework a piping system, we have to disconnect all the pieces of equipment: The boiler, the pumps, the users, whatever it might be, and isolate those. In order to do that, we have to remove this insulation from all these flanges or unions that are used to connect all these different users.
Q I thought I saw at one point in the video we watched that insulation isn't put on the unions. Has that been the case in your experience?
A It depends. There is times when unions are not covered, but there's a lot of times when they're not (sic). If they're in a personnel area where there are actual people working close to them and could get burnt, they would be covered for personnel protection. If they're in an isolated area where personnel cannot get near them, chances are they may not be. They might be left open.
Q And you just mentioned flanges. Is that an example

of a flange you might see on --
A That is a typical flange. That's a four inch. You can tell, it's got eight bolt holes all the way around it. There are different size flanges. They range all the way up to 24 inch. 48 inch. They get pretty huge. And the bigger the flange, the more bolt holes you have around it. The higher the temperature, the higher the pressure, the thicker the flange might be. This one here is probably about three-quarters of an inch thick. Those flanges can run up to three to four inches thick, depending on how many pressures you're working with.
Q It looks to me to be a two inch. Is this --
A That's a two inch, yes.
Q -- a typical example of a two-inch flange?
A Right. That's just for about a 150-pound system. If it was a higher pressure, those flanges would be much thicker and much bigger.
Q Say temperature 400 and above, how big would the flanges be?
A Those flanges would probably be close to an inch-and-a-half thick. The diameter would be bigger, the center hole would stay the same, and the bolts would be larger.
Q Okay. The diameter --
A The diameter of the pipe would stay the same.

Q Right.
A But the flange would be bigger because you can have more bolts to cover, to tighten it to make sure that's a good seal.
Q And how physically do you get the insulation off of the flanges?
A Well, us in the open construction area which I worked, I did not work in-house a lot of places, we would just knock it off with a hammer, hacksaw, anything we could find because we were not interested in salvaging the parts. We just broke it off and let it drop on the floor.
A lot of factories we used to go into for a shutdown or a turnaround, they might have their in-house people remove the pipe covering because they would like to save the pieces so they could reuse them. Other than that, we would take it off.
Q So was that a common practice that at some facilities they would try to salvage the insulation and reuse it instead of using new insulation?
A That's correct.
Q Now I think you had talked about expansion joints. Is this an example of an expansion joint?
A Yes, sir, it is.
Q Is any kind of testing or maintenance performed on

an expansion joint?

A On a high temperature hot water system, depending upon the pressure that's in there, periodically they have to check the wall thicknesses on the pipe which means they have to take in what they call -- it's an ultrasound to tell the thicknesses on the pipe. Because the pressure, volume and velocity of the water going through these things wears, just like it would wear rocks on a beach. See how smooth they get? Same thing happens inside that pipe.

So very possibly a high pressure system like that, they might want to check the thicknesses on that pipe maybe every five years, maybe eight years, depends upon what engineering tells you to do. Then you have to go in and take the insulation off. And the vital places where you would test would be where the curves are. That's where most of your wear would be on the pipe; to check to make sure the thicknesses were not getting down below what engineering figured was a safe place.

Q And Mr. Ferriter, I think you mentioned something called a *heat exchanger*. What is a heat exchanger?

A Well, the easiest way probably to describe a heat exchanger is if you take a coffee can and you drop, say, a handful of straws in it, and then you take and put a cover that's got a hole for each one of these straws and

put it on top of there. Now you've got water or product running through the straw, and on the side of the coffee can, you put in water around the straws, the outside of the straws.

So what happens is the water that's going in the coffee can on the side is the real hot water, the water that's going through the straws is cool water and you are warming it up so you can use it for another process. That's exactly what a heat exchanger is. It exchanges heat from one system to another.

Q And is testing and maintenances performed on heat exchangers?

A Most of those are pretested at the factories before they come out. If they -- they have labels on them on what their test pressures are. If their test pressures are above or the same as the test pressures that we're going to use in the pipe, we can test right through them. If their test pressure is lower than what we're going to test in the pipe, then we have to disconnect them, line them up, and test them separately.

Q Okay. And are heat exchangers insulated?

A Yes. They're insulated with block insulation.

Q Any cement?

A Yes.

Q And that has to be removed if you have to take it

out to test it?
A Yes, we do.
Q Now we talked a little bit about valves and I'm going to go back to the original drawing we had. I think you mentioned up in the right-hand corner that's a relief valve?
A Yes. Those are relief valves.
Q And what is the purpose of a relief valve?
A The purpose of a relief valve is to make sure that the system does not get overpressurized or overheated. And it's vented through the atmosphere out of the building. So that is the main safety that's on that system. And there might be numerous relief valves on that system. Any time that any part of the system can be isolated by valves, there's going to be relief valves in between there. They're all piped to the outside for personnel protection, protection of equipment, everything else. The main thing is for personnel protection.
Q And would that be an example of what a relief valve looks like?
A Yes. That's a relief valve right there. Some of them are very big; some of them are huge; some of them are as big as a six-foot man. It depends upon the size of the system you want to talk about.

Q Really. There's one as tall as me.
A There's one as tall as you. In fact, I bet you there's a few taller than you. There's some pretty good size. It depends upon -- on a nuclear powerhouse, they're probably about eight foot tall and they probably have maybe a 48 or maybe even a 60-inch relief going out the roof. Because it's so much volume that's in that pipe that's in there, they have to have a big enough vent to get that stuff out fast enough to relieve it.
Q And how do you test them?
A We send those out to an independent test lab to have them tested.
Q Are the valves insulated?
A The valves are insulated.
Q With block and cement?
A Yes, sir.
Q So, in order to remove them and send them out for testing, you have to remove the insulation?
A That's correct.
Q And what other kinds of valves other than relief valves do we see on this?
A The main valves are shut-off valves and these are either open or closed. I believe we got a picture of one of them there.
That's a picture of a regular gate valve. That

gate valve is either open or closed. You see the stem up on top here, that stem rises. It's a screw. And as you turn that handle, that stem will come up to show you that valve is open. When the stem is down in the position it is right now, that valve is closed.
Q And how do you perform testing and maintenance on a shut-off valve?
A Usually we open them up and test right through them. We put in what you call *blinds*. We put a blind on this flange right here -- this isn't working too good. We use a blind flange in there, which is a blind piece of metal with no hole on it, and that's what we test against.
Q And in order to perform that testing, do you have to remove insulation?
A Yes, sir.
Q And is that block and cement insulation?
A Yes, sir.
Q Any other types of valves depicted on this diagram?
A Yes, there is. There's usually control valves, this right here, on all your users. All your users will have a control valve on it and that is operated by a thermostat or possibly a aquastat or a different kind of control that lets them know what kind of temperature, water they want to do this process, and that valve will

modulate, open and close as water is needed in the system.

Q And are those tested?

A Yes, they are. Those are usually tested by an independent firm, too. We usually send those out.

Q To remove them and send them out for testing, do you have to remove insulation?

A Yes, sir, we do.

Q And that would be block and cement insulation?

A Yes, sir.

Q We talked about shutdowns and scheduled maintenance. Are there any times independent of shutdowns and scheduled maintenance when pipefitters such as yourself have to perform repairs on high temperature water systems?

A Yes, there are.

Q And could you describe for us what those circumstances would be?

A Yes. Say on this particular outfit here, say we had a pump went bad or a pump was having troubles, it was making noise and they wanted to check on it. Now if they had a outage or a shutdown coming up, say, June 1st, and say this pump, started having trouble with it on April 1st, they might move that shutdown up so they could coordinate it with taking that pump out. Instead

of shutting down twice, they'd do it all at one time. Then we would go in and work on the pump and do the other maintenance as we go along.

Q So as a matter of good economic sense if you have to shut down a part of the line because a piece of equipment goes bad, might as well get the rest of the maintenance done while you're shut down?

A That's correct.

Q What is a thermal shock?

A A thermal shock is mainly when hot meets cold. Probably the best description of thermal shock is in a thunderstorm, when you get a loud bolt of lightning and a real clash of thunder, what that is, that's cold air meeting hot air. The louder the thunder, the bigger the temperature difference. That's mainly what a thermal shock is.

Q And do thermal shocks occur on high temperature water systems?

A Yes. Quite frequently.

Q And can you tell us how and can you tell us why?

A A lot of times it's usually a control valve that might not work right or fail and it might let hot water into a cold vessel or a vessel containing cold water, and when they hit, it causes quite a catastrophe. It can break fittings. It can break pipe. It can cause a

lot of damage.

On other times, it could be human error. It could be somebody opening a wrong valve. Maybe he decided to drain the system without checking something that he should have done before. There's a few different deals, but mainly it's a mechanical deal where something has failed in a control valve or a control system of some kind.

Q So are these high temperature water systems kind of volatile, dangerous?

A Very. They're probably the most dangerous system we work on outside of the chemicals and -- hydrochemicals and hydroflammable stuff in refineries. The high pressure out-water system is probably one of the most dangerous.

Q And why are they so dangerous?

A Well, what happens is water that's heated to that extent expands so fast when it hits the atmosphere. If you took a ten-gallon bottle of water and heated that thing up to 400 degrees and then pull the cork on it, that thing would expand 10 times to 100 gallons right now within seconds. If you done it in this room with a 55-gallon barrel, it would fill this room in seconds and I would say get out of here. That's how volatile that water is because it flashes right now into steam being

under that high pressure and high temperature.

MR. HANBURY: Your Honor, can I have one moment?

THE COURT: You may.

(Pause at 2:35 p.m.)

Q Going back to the flanges just for a quick minute, what part of the flanges, if any part, are covered with block or cement insulation?

A The whole flange from this area down is covered. The pipe insulation will just come up about to where that well connection is right there. Everything else would be block, cement.

Q Okay. These holes in the flanges are for bolts?

A Yes.

Q So two flanges can be bolted together?

A That's correct.

Q And they're covered with block and cement as insulation?

A Most of the time on high pressure hot water, yes.

Q So to remove that insulation, you would need to remove it from the bolts and --

A And possibly back six to eight inches back on the pipe so you could pull the bolts out, depending upon the thickness of the covering.

Q How often is testing done on a high temperature

water system with respect to control valves?
A That's usually set up with the scheduled shutdowns. The engineering and architects usually take and say the span of that control valve working properly probably would be one year. So they'll schedule their shutdowns for one year and they will do all their repair work on control valves, RVs, no matter what it is during that shutdown.
Q Taking our relief valve again, how often are these tested?
A These are tested every shutdown.
Q Every year?
A Every year. It depends upon what the insurance company calls for on that test valve. All high pressure hot water lines and steam lines are regulated by the insurance company. The insurance company tells you when you have to take and test those relief valves and it's usually on a yearly basis or maybe a two-year basis, depending upon the pressures and temperatures you're working with.
Q Because they're such a vital part of the system?
A They are the vital -- they're the actual safety on that system that might save a lot of people or a lot of equipment or whatever.
Q And referring to this photograph, explain for the

jury where the insulation is on this.
A The insulation would usually come up around this area right here. The top of this valve will be exposed because sometimes they have an operating lever on there and you can tell if that valve is leaking through a little bit; if that lever is not where it's supposed to be.
This whole part of this valve down here will be covered. This exhaust pipe that goes out through the roof to the atmosphere is always covered.
Q Now on all of the valves we've talked about where you, as a pipefitter, have to remove insulation, when you remove that insulation is there any residue of the insulation remaining on the valve?
A Yes, there always is.
Q Describe it for us.
A Well, usually what happens when you've got high temperature systems, it's so hot it bakes some of that insulation and insulation actually adheres right to the metal. It's -- it gets caked on there pretty darn hard and you really have to work at it to get it off. It's quite prevalent that you see a lot of pieces stuck on there.
Q And it's your job as a pipefitter to remove it?
A If it's in our way, yes. If it's not in our way,

we can leave it there. But most of the time it's in our way and it's hampering our progress. We have to remove it, yes.

Q And with respect to the shut-off valve we've already talked about, can you describe for the jury where the insulation is on this?

A The insulation usually comes up again to this flange. It's a removable top, so that we always make sure that the operating system is working. You can view this screw in between these two pieces of metal right here and you can tell if that valve is open/closed by looking at the top of it.

And also you have a packing system right around here that seals that stem that goes down from the product that's inside so you can check and see if that valve is leaking.

Q Now control valve, does that appear similar to a shut --

A The control valve is very similar. The only thing we'd do is we'd remove this head here, this part up here, and we'd replace that with a head that's got a pneumatic operator or maybe possibly an electric motor on it. That's the only difference though. That's the basic problem. There's so many different kinds and configurations it would be confusing, but the basic type

is just what's on top of this valve.
Q And the control valve would be insulated the same way?
A Very similar. Up to this bolt circle, yes.
Q What is a check valve?
A A check valve is nothing but a little gate that hangs in the water. It's got a seal on it, and as long as the water is flowing, this gate is up or flapping possibly. When this water shuts off or the pump shuts off, this gate swings down. It seals so that no water can come back in and reverse the impellers on the pumps really is what the problem is because you can break the impellers on the pumps by the water rushing back in. So a safe valve or a check valve is really a safety for pumps for the water flow.
Q To prevent back flow of --
A To prevent back flow, that's correct.
Q And how often are those inspected?
A Those are basically inspected as you go along or during a shutdown. You can usually tell because they will start clattering. If the gate is getting loose in there or something during an operation, you can tell if you stand next to the pump, you can hear it making noise. But mainly a lot of those valves will last for years without really having a problem.

Q Okay. I think I'm gathering from what you've told us on all of these valves, insulation is removed just for the purpose of getting access to the valve.
A That is correct.
Q And what type of tools do you use to remove it?
A We use anything we have. If we've got a hacksaw or a hammer or -- you name it. We knock it off just to get it out of our way because we don't want nothing to do with it.
Q What are the dimensions of the insulated part of the valves on six-inch lines?
A That depends upon the temperature in the line. It could range from an inch to two inches, maybe up to four inches.
Q Okay.
A The temperature really controls what the thickness of the covering would be.
Q Okay. So you would look to the temperature of the line?
A Well, actually the engineer would state what size covering has to go on what size lines, depending upon the temperatures.
Q Sir, are you being compensated for your time here today?
A Yes, sir, I am.

Q How much are you being paid?
A $50 an hour. The going rate for a pipefitter.
Q And is that how you arrived at $50 an hour; that it's what you would have been making as a pipefitter?
A That's correct, sir.
Q And have you worked on any other cases with my law firm?
A Yes, sir. I've given, I believe, five or six depositions.
Q In other cases prior to this one?
A In other cases, yes, sir.

MR. HANBURY: That's all I have. Thank you, Your Honor.

THE COURT: Very well. For break purposes, about how long do you think the cross will be?

MR. MOORE: Mr. Feldmann is going to do it.

THE COURT: Ballpark.

MR. FELDMANN: Probably half hour, 40 minutes.

THE COURT: You guys want your break now or you want to wait? Now. All right. Let's come back at about three o'clock. Okay?

(Jury excused from courtroom at 2:46 p.m.)

THE COURT: All right. Everyone please be seated. Mr. Ferriter, you're free to take a break, too, but I just want to remind you that technically you're

still on the stand during the break. Obviously you're not, but the point is you cannot talk to anybody on your lawyer's team or to Mr. Bushmaker while you're on break. Okay?

THE WITNESS: I just wanted to stretch my legs a little bit.

THE COURT: You can leave the room if you want. I'm just saying don't talk to anyone.

THE WITNESS: That's fine. I'm just stretching my legs.

MR. MCCOY: I'll give you that big wrench and you can work on that for awhile.

THE WITNESS: No, I've had enough of that.

THE COURT: All right. Did anyone have anything for the Court before we take our break? Mr. McCoy, what have we got after Mr. Ferriter?

MR. MCCOY: Let's see, we have two -- no, we have one read-in deposition. It's very short. I don't think it's more than 15 or 20 minutes.

THE COURT: And then we've reached the end of today based on the Court's ruling this morning?

MR. MCCOY: Yes.

THE COURT: Okay. Fine.

MR. MCCOY: Yes. I was going to show at the break -- maybe we could have agreement on two exhibits

from Mr. Mueller's deposition.

THE COURT: That's fine. That's fine. And when we're done with Mr. Ferriter, we're done with your read-in, we'll talk about what happens tomorrow and Friday. So with that, you guys take your break, too. We'll be back at three.

(Recess 2:47-3:00 p.m.)

THE COURT: All right. Mr. Ferriter, why don't you come back and just stay standing. Let's bring in the jury, please.

(Jury brought in courtroom at 3:02 p.m.)

THE COURT: Everyone please be seated. Are we ready for cross?

MR. FELDMANN: Yes, Your Honor.

THE COURT: Let's begin.

CROSS-EXAMINATION

BY MR. FELDMANN:

Q Good afternoon, Mr. Ferriter.

A Good afternoon, sir.

Q I don't have a lot of questions for you, but there are some things I do want to cover with you. We had an opportunity to see your resume before today and I had a chance to look through it. You were in a local that was based out of Chicago?

A That's correct, sir.

Q You were never in the local that Mr. Bushmaker was in; correct?
A No, sir.
Q Have you ever been to Wisconsin Rapids?
A No, sir.
Q Okay. So you never have been at any of the Consolidated paper mills in Rapids; correct?
A That's correct, sir.
Q And you haven't been to the Consoweld -- either one of them, the old one or the new one; you haven't been in any of those places?
A No, sir, I have not.
Q So you haven't had an opportunity to see the systems and the piping or whatever might be in the plants that are owned by Consolidated Papers; true?
A That's correct, sir.
Q And as far as the interworkings and hidden mechanisms of those plants, so to speak, that's something you have no personal knowledge of; correct?
A That's correct, sir.
Q Okay. I want to -- you wrote a report in this case; correct?
A No, I did not, sir.
Q Okay. Because we were given a report that looks like it has a signature on it over a Joseph Ferriter.

That is you?
A That report was written for a terminus, I believe.
Q Well, it covers a lot of things.
A Yes, it does.
Q And it was actually filed in this case. Were you aware of that?
A Yes, I was. I'm sorry. I should have realized that was the document you were talking about. I didn't know that was the document you were talking about, sir.
Q This one is dated October 18, 2011?
A That's correct.
Q Okay. And information that you contained or put in this report was some information based upon your experience?
A That's correct.
Q And the information that you put in this report is true and accurate to the best of your knowledge; correct?
A That's correct, sir.
Q Okay. We'll get back to that in a minute. I'd like to talk to you about a few of the items that you talked to the jury about today, and more particularly, I'd like to talk to you about connections. One of the connections that you talked about was a thing called a *flange*.

A That's correct.
Q I grew up in a rural area, so bear with me. I'm not schooled in all of the things that you were talking about today, so I'm going to have you maybe do a little bit more explaining for me. But other things that you talked about, there were a number of different valves; is that correct?
A That's correct.
Q A flange, as I understand it, is where there's a connection between the ends of two pieces of pipe. Sometimes you have a flange connection and sometimes you connect them in other ways; correct?
A Right. Mainly on the systems we're talking about, it's flanged or welded.
Q Okay. All right. And when you have a flange connection, is that what this is?
A That's correct, sir.
Q So it's actually two pieces of metal that come together and form some sort of a intersection of the two pipes.
A That's correct, sir.
Q And you draw together using these bolts.
A That's correct, sir.
Q And is there a material that's used between the surfaces of the flange, the two pieces of flange in

order to seal the --
A Yes. It's called --
Q -- space between them?
A Yes. It's called a *gasket*, sir.
Q Okay. And -- all right. So you have gasket material that you put in between there. And is that a material that you can, in the 50s and 60s, you would buy from some sort of manufacturer of gasket material?
A Yes. We'd buy it from a piping supply house.
Q Okay. And was it part of your job as a pipefitter to use gasket material?
A Yes, sir.
Q Okay. And it was something that you did frequently; correct?
A That's correct, sir.
Q Any time you were going to work on flanges like this and you had to separate them, would you replace the gasket material?
A Yes, sir.
Q All right. And how would you go about doing that?
A Most of the gaskets that were available to us were preformed, precut from a manufacturer or supply house, whichever you say. At times we would have to cut our gaskets out of a sheet material, but that was very rare.
Q Okay. And if you were working with a flange --

with flanges that were on, say, a high temperature water system where the temperature, the operating temperatures were up at 400 degrees Fahrenheit the way you were telling us before, would the material that the gaskets were made out of be asbestos-containing back in the 50s and 60s?
A Yes, sir.
Q And when you would remove that gasket material, the asbestos gasket material, sometimes would there be a residue that you also had to also clean off of the flange surfaces?
A Yes, sir.
Q And, you know, I always think of it kind of like when we buy a glass at Target and they've got those labels on; you try to peel them off. You get this residue on that; either you break your fingernail on trying to get it off or you've got to use Glue Gone or whatever they call that stuff to get it off. Is that kind of the same principle?
A Same principle, yes, sir.
Q Doesn't all come off easily. You have to get it off using other means.
A Usually scrapers.
Q Okay. Sometimes wire brush?
A Wire brush, yes.

Q And sometimes you use a power wire brush if you can get it in --
A Very seldom you use a power wire brush because you could get carried away and put a little pressure on there and you could ruin the serrations on the flange space, which you don't want to do.
Q When you do that, when you're removing the gaskets from these flanges, that can create dust; correct?
A Yes, sir.
Q And if you're the pipefitter, if you don't have protection on, you inhale the dust; correct?
A That's correct, sir.
Q And flanges like that would be common that you would be doing maintenance and work around them during shutdowns?
A Yes, sir.
Q Would there be other times other than shutdowns where you would have to access the flanges and change the gaskets out?
A No, sir. If we were doing anything but rehab work, we would be using new gaskets and new flanges, which wouldn't have any residue on them.
Q Can these things leak at times?
A Yes. But very seldom if they're put together right.

Q If they do leak, then you have to go in and change out the gasket material?
A That's correct, sir.
Q Okay. You talked about -- and these flanges, this one is -- did you say it was two inch?
A That's a two-inch screwed flange, yes, sir.
Q When you say *screwed flanges*, are there different kinds of --
A Yes. There's screwed flanges. There's lap joint flanges. There's welded flanges. That's many types, but they all do basically the same job.
Q All right. All right. And I take it there's other sizes other than just a two inch.
A Yes.
Q It goes as big as the pipe.
A That's correct, sir.
Q All right. So if you have a six-inch pipe like that one over there and you had flange connections, you'd have, you know, a six-inch or an eight-inch type like this except a lot bigger.
A That's correct.
Q A gasket would be a lot bigger.
A Yes, sir.
Q You indicated before that sometimes the gaskets come from a supply house or from a manufacturer and they

may be already precut so you don't have to actually cut them to size; correct?
A That's correct, sir.
Q And other times you'd get sheets of this gasket material and then you have to cut it to fit the particular application.
A That's correct, sir.
Q Okay. When you cut that, that also creates dust; correct?
A Yes, sir.
Q And that can -- you also inhale if you aren't wearing proper protection; correct?
A That's correct, sir.
Q And this has been -- this was the practice back in the 40s and 50s and 60s; correct?
A That's correct, sir.
Q Okay. Now with regard to -- there was another thing. Valve. I've got one on the screen right now.
A Okay.
Q Is this a gate valve?
A That's basically a gate valve, yes, sir.
Q Okay. I got it right. I notice that there are -- there's a location in the gate valve right in the middle there where I'm pointing. It looks like it's bolted together there?

A Yes, sir.
Q Is that another location where you have to have -- where there's two surfaces that come together where you have to have some sort of a packing or a gasket material?
A That's correct, sir.
Q And when you would do a shutdown and you do work on the valves, would that be something that you would be replacing?
A Very seldom we would touch those bolts there. Unless we knew there was a problem with that valve, then we might have to open it to see if there was a problem, that the valve wasn't seeding right or something. But as long as the valve held during operations, we would have no reason to break that flange open.
Q Okay. So sometimes you might have to --
A Sometimes we would have to, yes.
Q -- if there appeared to be a problem or you were checking a problem or doing a test on it; correct?
A That's correct, sir.
Q Okay. And then there's also on this valve, there's on both like where I'm pointing right now, there's another location on each end there looks like a surface that's got holes in it. What would attach to those locations?

A A flange that was probably welded or connected to another piece of pipe.
Q Okay. So would those be other surfaces on the valve that if you remove the valve, you'd have either some sort of a gasket or packing material that you'd have to use?
A Yes, sir.
Q Okay. So, same process with a flange, you'd have to clean off any residue from the gasket -- let me ask you this first because I noticed in your report you refer to a couple of different types of products. One was called *rope packing*?
A Rope packing is packing that goes up in this part of the valve, up in here, to seal the stem from the product that's inside the pipe.
Q Okay. And then the rope packing wouldn't be used on these flat surfaces.
A No, sir.
Q That would be where you put the gaskets.
A That's correct, sir.
Q So each time that you would do valve maintenance, you would essentially remove the gasket material where the flanges came in and then replace the gaskets when you put it back together; correct?
A That's correct, sir.

Q And so once again, that's a dusty process because you have to remove the gasket material, you have to replace it or perhaps even cut pieces to fit and then put it back together again, and during that process, you're creating dust which you would also inhale if you're working on it right there on the bench or wherever you work on them as you do that maintenance; correct?
A Yes, sir.
Q Okay. And that would be something that you would do on shutdowns; correct?
A That's correct, sir.
Q Okay. You said that this rope packing material -- we haven't talked about that. What is that?
A Rope packing material is a -- just like it explains, it's a piece of rope and it's usually -- they come in circular little sections and they have a connecting place. The circle is not complete. It's an open circle that you can open. You drop those little sections in around that stem that's on there and you stagger the split and that tightens down and makes a seal around the stem so the product cannot leak out through there.
Q Okay.
A That's what rope packing is.

Q Are there other places you use this rope packing other than the stem on one of these valves?
A It could be used on the inspection plate on a boiler, on inspection plates on a vessel. But a lot of those have pre-formed gaskets, too. Very seldom you'll see rope packing on them, but you do see it once in a while.
Q Okay. But you as a pipefitter or Mr. Bushmaker as a pipefitter just generically, pipefitters deal with rope packing on a regular basis?
A Yes. Not on a real regular basis, but we do deal with it often.
Q And the rope parking that was used on high temperature water systems back greater than 400 degrees Fahrenheit or 400 degrees Fahrenheit back in the 50s and 60s, that was asbestos material?
A I would say yes, sir.
Q And from time to time as a pipefitter you'd have to remove rope packing material as well; correct?
A That's correct.
Q And that's because -- well, like anything else, it has a finite life; true?
A That's right. And especially under high temperature it gets brittle.
Q That's what I was going to ask you. What happens

to it as it ages?
A It gets brittle and loses its resiliency.
Q Okay. And so when you need to remove it, that also may create dust because it's kind of decomposed to the point where it's brittle and breaks apart; correct?
A You're correct, sir.
Q And that would be another occasion where a pipefitter doing that kind of work may inhale dust from a product with asbestos in it; is that true?
A That's correct.
Q All right. Now in a hot water treatment system, we saw this drawing that you made, and thank you. The system that you've kind of drawn with this diagram, I take it there are just scads of these valves in the system; correct?
A That is correct, sir.
Q I mean between the pump and the boiler and all the user equipment and the accumulator and heat exchanger and all that, you've got valves everywhere; correct?
A That's correct, sir.
Q Okay. So when you do a shutdown and you have to either test those valves or do maintenance on them or replace the gasket materials, potentially you've got a lot of work to do every time on a yearly basis to do those valves; true?

A That's correct, sir.

Q Okay. And I think that you indicated -- I just said yearly, but I think you told us that that's normal in an industrial setting with valves like you've described, it's normal to maintain them on a yearly basis during shutdown; correct?

A That's correct, sir.

Q There was another -- another valve that you talked about.

MR. FELDMANN: He's worse at this than I am.

MR. MOORE: My eyes are worse.

THE COURT: Takes the paralegal to get it; right?

MR. MOORE: That's the truth.

MS. BENSON: Well, don't give me too much credit. We're not there yet.

MR. MOORE: Mr. Ferriter did better with erasing the drawings.

BY MR. FELDMANN:

Q This was another valve that you talked about before and I'm trying to remember what this one was called.

A Called a *relief valve*.

Q Relief valve. That's the RV that's on that drawing. Okay. So the relief valves also have connections where they connect to piping?

A That's correct, sir.
Q And as you stated before, even though when we first looked at this I thought all relief valves were this big, you said that they could be as big as six foot or eight foot or bigger.
A That's correct, sir.
Q So it just depends on the system, what they're attached to and so forth. But they also would have places where they would connect and you'd have to put either -- you'd have to put some sort of insulation between the connections to make them water tight?
A Insulation, sir?
Q Yeah. Like a gasket.
A A gasket, yes. Not insulation. Yes.
Q Right. But back in the 50s and 60s, the gaskets that were used with these as well, as long as the water temperature was high enough, they were asbestos-containing; correct?
A That's correct, sir.
Q Okay. And those gaskets, like the ones we talked that were used on the gate valve that we looked at before, the same kind of stuff, you'd either have to buy them that were all precut or you would have to cut them yourself; correct?
A That's correct, sir.

Q Okay. Those were all things that pipefitters did.
A Yes, sir.
Q Okay. And you said that there was a whole bunch of these on hot water systems; correct?
A Yes, sir.
Q Okay. And then this would be another valve and when you did shutdowns on a yearly basis, they had to be worked with. There had to be gasket material taken off of them; new gasket material supplied and put on them just like for the gate valves; correct?
A That's correct, sir.
Q Okay. Now we talked about relief valve. We talked about gate valve. Are there any other valves in this system -- he's getting there.
Are there any other valves in that system other than those two kinds of valves? I think you mentioned a user control valve.
A Control valves, sir, is the same as a gate valve only it has a motorized operator on it. That's it.
Q But they're in the system and they connect to the piping as well?
A That's correct.
Q So same thing with regard to the control valves. As with the gate valves and relief valves, they also are maintained during the shutdown and they would also have

to be taken out and put into the system, gasket material used to do that; correct?
A That's correct, sir.
Q Okay. In your report you mention union jurisdictional lines. What does that refer to?
A Union jurisdictional lines means the separation of whose work is whose between possibly the pipefitter and the boilermaker, which are very closely associated.
Q Okay.
A The pipefitter and plumber maybe, which are very closely associated. We all belong to the same international union, we just have different phases of piping that we do.
Q You mentioned pipe coverers during your testimony. Are pipe coverers in a different union than pipefitters?
A Yes.
Q When you talk about union jurisdictional lines, what you're talking about is that if I'm a pipefitter, I'm not going to do a boilermaker's job because that's his trade.
A That's correct, sir.
Q And in the trades back in the 1950s, 60s, 70s, probably all the way up to today, as far as you know until you retired were these jurisdictional lines followed by the trades?

A Yes, sir.
Q And as far as -- probably are today; correct?
A That's correct, sir.
Q In other words, don't do my job.
A There was verbal agreements where economically-wise it would not be smart to bring another trade on the job for an hour when we could do it ourselves. But we would not replace it. They would have to come and replace it or something like taking the covering off or maybe unbolting a flange, that belongs to the boilermaker. There was little verbal agreements on that. Because economically for the customer, it would cost him eight hours pay for them to get a boilermaker out when somebody could do it that was on the job.
Q If it was something --
A Minor.
Q -- minor --
A That's right.
Q -- you could step over the line just a little bit. But if it was something that was bigger, then the other trade was brought in and they did it.
A That's correct, sir.
Q So when you did your shutdowns and you're removing block insulation and insulation from some of these valves and so forth, usually what happened was those

weren't -- when it went to put stuff back on to reinsulate things, that was what the pipe coverers did.
A That's correct, sir.
Q Not the pipefitters.
A That's correct, sir.
Q Okay. And then correct me if I'm wrong, but when you have a valve or you have a flange and you want to get at it and you were talking about removal, about using a saw or a hammer or something like that, I take it that you're pretty much destroying the covering that was on there, the insulation that was on there; correct?
A That's correct, sir.
Q And so that was gone. And when it had to be replaced, then you'd have to bring in somebody -- the pipe coverer would come in and bring different material.
A That's correct, sir.
Q I just wanted to be certain the video that we saw that was done by the U.S. Government, that showed a fellow or men doing various types of work. And the work that they were doing was not the work that the pipefitter would do, that's what -- the work that a asbestos worker or a pipe coverer would do; correct?
A That is correct, sir.
Q Now I take it that as far as these high temperature water systems, for you to tell me how many valves or how

many flanges are in a hot water system, you actually have to go and see that system because you'd have to know the dimensions of the plant and how big the system is and what are all the equipment is that it runs and all that stuff; correct?
A Basically yes, sir.
Q Okay. So if I asked you -- there's not a generic heating system that you can go out and buy at Fleet Farm or something like that and put in one of these buildings. These are engineered systems; correct?
A They're engineered systems, but they're all of a generic plan. All the engineering does is maybe add to them or take from them, but basically they all operate the same.
Q Okay. But a high temp water system that would be in a paper mill in Wisconsin Rapids might be significantly different in size and scope than a hot water or high temperature water system that's at a Mobil Oil refinery; correct?
A In size and scope only. Other than that, it would be identical.
Q It would work the same way.
A That's correct.
Q Except there could be a ton more valves and flanges and so forth in the bigger system.

A That's correct, sir.
Q So you don't have any way of knowing how many valves or flanges had to be maintained at this paper mill in Wisconsin Rapids because you don't have enough information.
A That's correct, sir.
Q All right. I just have -- there's a few statements that I want to check on that you've made in your report. One of them says, "The removal of gaskets on all piping systems was done on all pipefitting remodel, repair, maintenance and rehab jobs before about 1988 without protective measures for asbestos and was a dusty process for lines or systems operating all operating pressures and temperatures. The same was true for removal of packing materials."
The packing materials you referred to are the rope packing material; correct?
A That's correct, sir.
Q And when you talk about rehab jobs, what is a rehab job?
A A rehab job could be a -- say a factory that maybe the one owner sold it to another owner. The new owner comes in. He might want to upgrade some stuff or possibly add things or change. That's what we call a rehab job.

Q Okay. I want to return to the packing material because I want to make sure that I understand. As far as utilization of packing material, would you use that type of material in applications such as in hydraulics or pneumatics?
A Basically it might not be identically the same, but any place that a shaft for possibly a valve or a pump would enter into a system that's carrying liquid or whatever process is in there, you would have some kind of packing in there. Whether it be made of the same material or maybe teflon or some other composite material, I have no idea. There would be something in there to seal that, yes.
Q Okay. And this packing material that it comes in a package that's like rope; right?
A That's correct, sir. Some of it's precut, some of it comes in random lengths.
Q It says in your report "Pipefitters install the packings in valves, shafts, drive shafts on pumps, circulating pumps, piston-driven pumps, other pumps, manway openings, inspection ports and burners, and other equipment. What's a manway opening?
A Manway is usually an inspection plate that's in a vessel or a boiler --
Q Okay.

A -- that you can open to go in and maybe check the tubes for thickness or something like that. Wear and tear on them.
Q Okay. Says here "Pipefitters remove the packing material using small cork screws, screwdrivers, and other tools." And it says, "Packing is hardened and lost resilience over time if used in high temperature above about 120 degrees."
A That's correct, sir.
Q I think we touched on that a little bit before. Okay. Then it says, "Gaskets are abundant in the pipefitting industry and where asbestos containing on any lines operating at temperatures above 120 degrees before 1988. Most equipment valves of all types: Pumps, tanks and specialty items come with flanged connections for ease of removal for replacement or maintenance. Pipefitters had to do gasket work on almost every job."
A That's correct, sir.
Q And then lastly it says, "On the installations of the sectional and package boilers..." I don't know what a sectional or a package boiler is.
A Sectional boiler is a boiler that has to be put together in place like in a basement of an apartment building or something. You bring it in in sections and

you have to build it actually on the job.
Q Okay. It says "On installations of those sectional..." and then it says package -- I take it a package boiler was all put together before.
A A package boiler is one piece that you pick up and set on a slab and piping to it.
Q Says "Pipefitters had to install gaskets and rope materials..." -- that would be the rope packaging?
A Yes, sir.
Q "...which were asbestos-containing before 1988. The work with the rope materials was dusty and sometimes the gaskets had to be cut. Removal of gaskets and rope materials during maintenance was done by pipefitters and was a dusty process."
A That's correct, sir.
MR. FELDMANN: Thank you, sir. That's all the questions I have. (3:30 p.m.)
THE COURT: Very well. Mr. Hanbury, would you like to redirect?
MR. HANBURY: Yes, Your Honor. Thanks. Just very briefly.
THE COURT: Take your time.

REDIRECT EXAMINATION

BY MR. HANBURY:
Q Mr. Ferriter, you were asked about residue of

gaskets on flanges. How thick was the gasket material on the flanges for a high temperatures system, on say a six-inch line?
A Most of them were around -- between an eighth and a quarter inch. The new gaskets that come out is Flexitallics; measure about a quarter-of-an-inch thick and they're compressible.
Q The Flexitallics weren't around in the 50s.
A They come out, I believe, in the 60s, I believe; in the middle 60s.
Q And what process creates more dust? Knocking off the block and cement insulation off of the components of the system or removing gasket residue from flanges?
A No comparison. It's block insulation.
Q For instance, the process of knocking block insulation and cement off of valves and other components, would the dust from that process be on your clothing?
A It would be on your clothing and be on everything around you.
Q Describe it.
A It would be like a light snow --
MR. FELDMANN: Sorry, Your Honor. This really goes beyond my cross.
THE COURT: No, it's fair game. I'll let it

go.

MR. HANBURY: Thank you.

BY MR. HANBURY:

Q Go ahead, sir.

A It could be like a light cover of snow on the ground around you. A lot of stuff would be just plain white around you if you worked in the area long enough.

Q And you were asked about inhaling the dust from gasket removal. Would you inhale the dust you just described, the light snow?

A Yes, sir.

Q Could you avoid inhaling it?

A Not really.

Q In the practice of knocking off block and cement insulation with saws or hammers or whatever tools you used, did that process continue up until the time you retired or -- and I'm talking about doing that without any ventilatory protection. Did that continue up to the time you retired or did that stop at some point?

A I'd say around the late 80s, around 1990 to be exact is when they come up with the asbestos removal, and a lot of the owners and contractors were aware of insulation material being in a lot of the buildings that we had to go in for shutdowns and stuff and they would hire asbestos-removal contractors to come in and remove

it.

Before that time, we used to do it ourselves or the pipe coverers, whoever was in that particular job that they were doing would have to remove the insulation themselves. But after the 90s, they had it pretty well controlled.

Q When you were describing union jurisdictional arrangements, I think it was like, for instance, when a particular job outside your jurisdiction would only take eight hours, you would have agreements where you would allow that sort of practice?

A Yes. I was talking mainly on like a new construction or a rebuild or something, not a shutdown, we had little agreements that we would do maybe an hour's worth of work rather than call in a pipe coverer, for economic reasons for the owners and everything else. But now a lot of times when we would go into a factory or possibly a industrial district, they might provide somebody to remove the insulation before we got there that was in-house.

Q In terms of those jurisdictional agreements, you were speaking in the context of yourself as a pipefitter working for a contract on a specific job; is that right?

A That's correct, yes.

Q Okay. It would be a different scenario for

somebody who is the employee of a manufacturing facility in-house?

A I believe so, yes.

MR. HANBURY: That's all I have, sir. Thank you.

MR. FELDMANN: Nothing further, Your Honor.

THE COURT: Any recross?

MR. FELDMANN: Nothing further, Judge.

THE COURT: Okay. Well, thank you, Mr. Ferriter. You're done. You're free to go about your business. Thank you.

THE WITNESS: Thank you.

THE COURT: Have a safe trip home.

(Witness excused at 3:35 p.m.)

THE COURT: Who's got the next witness?

MR. MCCOY: Next witness is another group of paper, Judge, so...

THE COURT: Okay. And for the court reporter's benefit, is this something where you can provide an accurate transcript later for our use?

MR. MCCOY: Yes, Judge.

THE COURT: Fair enough. Then who is being the witness today?

MR. MCCOY: We'll go back to Mr. Hanbury.

THE COURT: Very well.

MR. MOORE: What transcript are we doing?

THE COURT: We haven't heard yet.

MR. MCCOY: Right. This transcript is -- the name is John W. Humphrey. And that's H-u-m-p-h-r-e-y. His testimony was taken on May 23rd, 1979.

THE COURT: All right. Ladies and Gentlemen, I think you know the drill by now. Again, you're hearing sworn testimony from a previous deposition. This one from 1979. The testimony is evidence. The demeanor of our reader is not.

And Mr. McCoy, usual courtesy, please. When you switch pages, please announce it so counsel can follow along.

MR. MCCOY: Yes.

MR. MOORE: May I have a marked up copy, please?

MR. MCCOY: I thought we may have given you one copy of this this morning.

MR. MOORE: I didn't receive it this morning.

(Pause)

MR. MOORE: This one is not marked up, but I'll follow along.

MR. MCCOY: Do you want me -- I can do it straight from your page so you can see mine at the same time. How about that?

MR. MOORE: I can read over your shoulder. Can I do that, Your Honor?

THE COURT: If you're both comfortable with it, so is the Court.

MR. MCCOY: And this also includes, Judge, the questions that were chosen and answers by plaintiff as well as by defendant.

THE COURT: Understood.

MR. MCCOY: Joint effort anyway. Okay. So we're on page three.

MR. HANBURY: Yes.

(Deposition of John Humphrey read in 3:38-3:50 p.m.)

THE COURT: That's the end of the designations. Mr. McCoy, is this the juncture that we talked about this morning where we're done for the day?

MR. MCCOY: Yes. For today, Judge.

THE COURT: Understood.

MR. MCCOY: We've got more tomorrow.

THE COURT: Okay. Well, Ladies and Gentlemen, by virtue of calendaring, you're done for today. We're going to start with you again tomorrow morning at nine o'clock. Expect a full day tomorrow, probably until five, and we'll just take it from there.

So with that, you're excused for the afternoon.

(Jury excused from courtroom at 3:50 p.m.)

THE COURT: All right. Everyone please be seated. I'd like a pulse and temperature check or perhaps a road map as to what's left for the plaintiff and your anticipation of how we get there from here.

MR. MCCOY: What's left, Judge, is going to be Dr. Arthur Frank tomorrow. He's coming in from Philadelphia. He's a professor at Drexel University that specializes just as much, if not more so, than Dr. Brody in asbestos diseases.

THE COURT: Okay.

MR. MCCOY: And he'll be testifying. Another international personality. And then we have the testimony of Dr. Carl Bedrossian, which was the videotape deposition that we worked everything out on.

THE COURT: Okay.

MR. MCCOY: That will be played. I think that's a little over three hours. I expect pretty much tomorrow will be Dr. Frank and Dr. Bedrossian.

THE COURT: Okay. Well then what's left for Friday?

MR. MCCOY: Then what's left for Friday would be Gerald Bushmaker.

THE COURT: Okay. Well, let me ask you this: I don't know how long your two witnesses will take, but if it's three hours each, we've got six hours of trial.

That leaves at least an hour or two. Is there any problem with starting Mr. Bushmaker tomorrow afternoon if your other witnesses are done before four?

MR. MCCOY: I guess there's not a problem.

THE COURT: Okay. Well, and I don't want to catch Mr. Bushmaker by surprise. I want him to be aware that he may start his testimony tomorrow, depending on how other things work out.

Okay. So let's assume that Mr. Bushmaker either starts Friday morning or perhaps begins on Thursday afternoon and finishes on Friday. About how long do you think that will take? About when on Friday do you anticipate being done? Ballpark.

MR. MCCOY: I really can't say that because I don't know how fast Mr. Bushmaker will go from my end because he's not the professional speaker that we've had so far in this courtroom. But --

THE COURT: No, I understand.

MR. MCCOY: -- I know he's a little -- he's just not as quick. And also I don't know how long the defense cross will go. I understand that will take awhile --

THE COURT: Sure. And I'm not looking for precision.

MR. MCCOY: It's going to --

THE COURT: No, wait, wait, wait. Let me finish my question. Is it fair to predict that you will rest before the end of the day on Friday and that the defense team should be ready to start presenting evidence? Is that a likely possibility?

MR. MCCOY: I think it's possible that that could happen, but we have one witness who we can only get in on Monday. I think we both have a witness on Monday. These are experts that have been prescheduled.

THE COURT: Sure. But -- okay. I'll stop that thought and let's keep going.

MR. MCCOY: The expert --

THE COURT: So who's your expert Monday?

MR. MCCOY: The expert William Eweing, the industrial hygienist coming from Atlanta. He will not be as long as the others.

THE COURT: Okay. Well, again give me a ballpark estimate in terms of number of hours you predict. Just a prediction.

MR. MCCOY: For Mr. Eweing? His testimony on direct will be under an hour and a half.

THE COURT: Okay. Well then we've got the whole -- and at that point are you resting?

MR. MCCOY: That would be it.

THE COURT: Okay. Then we've got the defense

case. I know you guys have some experts. Just one? One witness?

MR. MOORE: Yes, sir.

THE COURT: For Monday?

MR. MOORE: Yes, sir.

THE COURT: Okay. Well, I'm not going to put you guys to your closings on Monday no matter what at this point, unless you all are predicting you'll be done by noon. Is that -- will we have a better idea on Friday?

MR. MOORE: Yeah, I think so.

THE COURT: Let's talk again on Friday then. I mean this all sounds doable. What I wanted to avoid and what you're telling me, what I'm going to hold you to is that the jury will be deliberating by Tuesday at the latest.

MR. MOORE: Absolutely.

MR. MCCOY: Right.

THE COURT: Might be deliberating Monday afternoon. We don't know. And again, I'm just looking for a range here. So what you're telling me is acceptable to the Court, but I just wanted to make sure that we were all still pretty much on the same page and it sounds like we are.

I presume that when the plaintiff rests, the

defense team will at least want to make its motion. I don't know that we'll entertain argument at that time, but obviously that won't be something that will delay us long.

So I don't know that we've got anything else from the Court's perspective. Mr. McCoy, anything else?

MR. MCCOY: I have a couple more comments --

THE COURT: Sure.

MR. MCCOY: -- about the next start of the proceedings going forward. One is we do have some additional exhibits, and the other one is the -- I think we have -- we'll have a resolution on the stipulation about the Rapid liability one way or the other tomorrow.

THE COURT: Sure.

MR. MCCOY: And I would also suggest that there are some questions on jury instructions that I think a lot of it is worked out, but there are some questions about those instructions which I suppose could be dealt with --

THE COURT: Sure.

MR. MCCOY: -- maybe on Friday or at the end of the day tomorrow. I think you need to hear some more testimony before you understand what the evidence is on that.

THE COURT: Let me suggest this, and I think

that this is a realistic suggestion based on what you're predicting, and of course things change. But I'd like to have a jury instruction conference with you guys Friday afternoon, okay? Because in the event that you're closing on Monday afternoon, I don't want you to get a final set of instructions from the Court the morning you close. That's not fair.

I want you guys to have the weekend to plan your closings. Now of course if you don't close until Tuesday, maybe we jumped the gun. But we may have a better sense of that Thursday night. But why Thursday is important is if we're going to meet on Friday afternoon, as is this Court's constant mantra, I'd like to keep it in front of me. I don't want to be catching up as we go.

So to the extent that the parties have reached agreements on what you think the instructions should look like, I'd like to see those by Thursday, close of business, whatever, so that if we do talk on Friday, which is what I'd like to do, I've seen them and can react to them and also make a record on those on Friday afternoon.

So Mr. McCoy, how does that sound so far?

MR. MCCOY: Fine. I think that's fine. I think we'll just confer with each other by email is easy

just --

THE COURT: Sure.

MR. MCCOY: We have no --

THE COURT: I don't know where you all are staying. Maybe you can find your favorite restaurant in Madison and parlay over a cheeseburger, but that's up to you guys.

MR. MCCOY: Which ones we have no objection to that are in the Court's proposal.

THE COURT: Sure. And let's also be clear: As is this Court's constant practice and as I promised you in this case, and I'm going to keep this promise, we're going to fine tune the instructions based on what came in. But I'm looking for guidance to the parties in that regard. I want you guys to tell me how you want them modified.

And in response to Mr. McCoy's concern, which I think has now abated in light of the Court's ruling on Motion in Limine Number 23, there was a concern that the jury instructions and the verdict form would change on the warning. I think that's gone, but we need to make a record on that at the appropriate time.

I haven't looked at the instructions since last week. I can look at them tomorrow if necessary. But I want the parties to get to the Court any substantive

changes and certainly any disagreements by close of business tomorrow on the instructions and the verdict form; at least flag them, even if you can't give me details, so that when we meet on Friday afternoon, we've got a general sense as to what's going to happen.

I'll give you a for instance. Thursday night you won't know with specificity what Mr. Bushmaker may say in terms of which products he's been exposed to, but I think you've got a pretty good idea. So of course I'd like what you think you're going to get presented to the Court Thursday afternoon, if possible.

Now, if we've got the whole weekend to work on it, I'm not going to make you jump the gun on that. I want your comfort level to be important -- to be accounted for here as well, but I don't want either side to go into the weekend blind. I want you to know generally and with as much specificity as we can muster on Friday afternoon what you're going to see from the Court in terms of both a verdict form and jury instructions so that you can plan your closings accordingly. Okay? So I'm trying to make it as easy and as useful for you as I can, but part of that means fronting stuff with the Court tomorrow if you can, and if not, at least flagging concerns so that we can discuss them on the record on Friday. Okay?

So Mr. McCoy, I may have interrupted you. Did you have other concerns you wanted to bring to the Court's attention?

MR. MCCOY: Let me think for one second, Judge.

MR. HANBURY: Your Honor, while Mr. McCoy looks, I just -- this isn't going to affect the time line you were talking about, but I did want to mention, because in the order of witnesses it wasn't conveyed to you the surgeon, Dr. Rashid, who performed Mr. Bushmaker's thoracotomy, he -- it looks like he's agreeing to come in Friday afternoon. He's got clinic Friday morning in Milwaukee and --

THE COURT: Have you got a subpoena on him?

MR. HANBURY: Yes.

MR. MCCOY: He's less than an hour though.

THE COURT: Well, either he has been served or he hasn't, but he defies a subpoena at his own peril and I'd like to think even a surgeon in Milwaukee will not defy a federal court subpoena. But I've been surprised before.

MR. MCCOY: I remember one instance --

THE COURT: Well, no, let's not go there.

MR. MCCOY: That was another case.

THE COURT: One final follow-up, Mr. McCoy. You mentioned that you've got other exhibits.

MR. MCCOY: Yes.

THE COURT: I would surmise as much. I'm not sure why you're telling me that. Other exhibits that are simply floating; for instance, like your worker's comp claims? Or simply exhibits that will be presented with witnesses who will lay foundations?

MR. MCCOY: Well, we're going to introduce the medical records.

THE COURT: Oh, well sure. But that's --

MR. MCCOY: Those are just going to be published. Not very many, but about -- I've got about six pages.

THE COURT: I don't need to know that. I expect that to be true.

MR. MCCOY: The next thing is we've got still a couple of these exhibits on notice which I talked about with Mr. Moore. One or two may require rulings, but we'll bring in the testimony to support foundation. That's brief.

The other thing was we do -- we are looking at the documents generated post-1960. As I said, there are some of those documents that talk about pre-1960 very specifically.

THE COURT: Right. And the Court has already ruled that within the limits of proving dangerousness of

asbestos, that's acceptable. We talked about that earlier today. The Court is prepared to offer a limiting instruction. The defendant's current position is that it would like to propose an instruction on that to be included in the jury instruction packet. So I think we're ahead of the curve on that one.

MR. MCCOY: So we've got an analysis today during the day, that James Hoey did, about which documents are in that category, and then I'll talk to Mr. McCoy about which ones we might offer.

THE COURT: Sure.

MR. MCCOY: But most of them were knocked out by the ruling. That's for sure.

THE COURT: Understood. Well, it's only four and I know you guys don't sleep anymore, so that gives you how many hours until 8:30 tomorrow? That's a lot of time. However, you will be breaking for that cheeseburger parlay.

But other than that, is there anything else that you wanted to put in the record this afternoon, Mr. McCoy, before I get the pulse and temperature check from the defense? If you need to consult with your colleagues, that's fine, too.

MR. MCCOY: I think that's it for now.

THE COURT: Mr. Moore, Mr. Feldmann, same

general question to you guys.

MR. MOORE: My pulse is fine.

THE COURT: Okay.

MR. FELDMANN: I still got one.

THE COURT: What the Court is proposing sounds doable? Realistic?

MR. MOORE: Yes. Yes, sir. Yeah.

THE COURT: All right. Well, then why don't we plan on our 8:30 start tomorrow just in case things come up, but I'm predicting it will be relatively short tomorrow in light of what I'm hearing tonight. But I would -- my comfort level is not going to allow me to say oh, just show up at nine.

MR. MOORE: We'll check your pulse, too, Your Honor.

THE COURT: That's a good idea as well. With that, we're done for today. Thank you all.

(Proceedings concluded at 4:04 p.m.)

* * * * *

I, LYNETTE SWENSON, Certified Realtime and Merit Reporter in and for the State of Wisconsin, certify that the foregoing is a true and accurate record of the proceedings held on the 6th day of March 2013 before the Honorable Stephen L. Crocker, Magistrate Judge for the Western District of Wisconsin, in my presence and reduced to writing in accordance with my stenographic notes made at said time and place.

Dated this 25th day of March 2013.

/s/_______________________________

Lynette Swenson, CRR, RMR, CBC
Federal Court Reporter

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